AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

NATIONAL LIBRARY OF MEDICINE
H M S

EDITORIAL BOARD

FRANK SPOONER CHURCHILL, Chicago

ABRAHAM JACOBI, New York City
DAVID M. COWIE, Ann Arbor, Mich.
JOHN LOVETT MORSE, Boston
EDWIN E. GRAHAM, Philadelphia

JOHN HOWLAND, Baltimore

VOLUME IV

1912

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

BACTERIOLOGICAL
LIBRARY
H M S

Um

CONTENTS OF VOLUME IV

JULY, 1912 NUMBER 1

PAGE

LEUKOCYTIC "INCLUSION BODIES" WITH SPECIAL REFERENCE TO SCARLET FEVER. JOHN A. KOLMER, M.D., PHILADELPHIA 1

THE CLINICAL SIGNIFICANCE OF ABNORMAL RESPIRATION AND DEGLUTITION CLICK OF FREMITUS: THE PNEUMONIC-RESPIRATION-PAUSE CYCLE. LAMBERT OTT, M.D., PHILADELPHIA 7

THE RELATIONSHIP BETWEEN THE INFECTION IN THE CHILD AND CLINICAL TUBERCULOSIS IN THE ADULT. FRANCIS MARION POTTENGER, A.M., M.D., LL.D., MONROVIA, CAL. 13

RECENT ADVANCES IN OUR KNOWLEDGE OF MEASLES. JOHN F. ANDERSON AND JOSEPH GOLDBERGER, WASHINGTON, D.C. 20

THE DIAGNOSTIC VALUE OF THE CUTANEOUS TUBERCULIN TEST OF V. PIRQUET. F. L. WACHENHEIM, M.D., NEW YORK 27

APPARATUS FOR COLLECTING INFANTS' BLOOD FOR THE WASSERMANN REACTION. KENNETH D. BLACKFAN, M.D., ST. LOUIS 33

TETANUS AS A COMPLICATION OF BURNS. CHARLES NEWBERGER, B.S., M.D., CHICAGO 35

AN UNUSUAL LESION OF THE RIGHT CRUS IN AN INFANT OF FOUR MONTHS. FREDERIC J. FARNELL, M.D., AND HENRY W. BURNETT, M.D., PROVIDENCE 43

STANDARDIZATION OF BLOOD-PRESSURE READINGS BY MEANS OF AN AUTOMATIC DEVICE FOR READING SYSTOLIC AND DIASTOLIC PRESSURES IN CHILDREN. B. RAYMOND HOOBLER, M.D., NEW YORK 46

TUBERCULOSIS OF THE MESENTERIC GLANDS IN INFANTS AND YOUNG CHILDREN. ITS EFFECT ON ABSORPTION. FRITZ B. TALBOT, M.D., BOSTON 49

PROGRESS IN PEDIATRICS—INDEX OF CURRENT PEDIATRIC LITERATURE 57

AUGUST, 1912. NUMBER 2

HISTORY OF THE CLASSIFICATION OF GASTRO-INTESTINAL DISEASES IN ANCIENT AND MODERN TIMES. ISAAC A. ABT, M.D., CHICAGO 65

STUDIES IN THE BACTERIOLOGY OF THE ACUTE INTESTINAL DISEASES OF INFANCY. BORDEN S. VEEDER, M.D., ST. LOUIS, AND ROBERT KILDUFFE AND O. T. DENNY, PHILADELPHIA 75

USE OF POLYCARBOHYDRATES IN THE DIET OF THE YOUNG INFANT. JULES M. BRADY, B.S., M.D., ST. LOUIS 89

APPENDICITIS IN CHILDHOOD. RUSSELL S. FOWLER, M.D., BROOKLYN, N. Y. ... 97

PROGRESS IN PEDIATRICS:

Abstract: Infant Feeding and Disorders of the Digestive System: Résumé of the Literature of the Past Six Months. Maurice Ostheimer, M.D., Philadelphia 104

INDEX OF CURRENT PEDIATRIC LITERATURE 121

SEPTEMBER, 1912. NUMBER 3

SOME FUNDAMENTAL PRINCIPLES IN STUDYING INFANT METABOLISM. FRANCIS G. BENEDICT, PH.D., AND FRITZ B. TALBOT, M.D., BOSTON 129

CONGENITAL OBSTRUCTION OF THE POSTERIOR URETHRA: REPORT OF A CASE IN A BOY AGED FIVE YEARS. J. H. MASON KNOX, JR., M.D., AND T. P. SPRUNT, M.D., BALTIMORE 137

290178

PAGE

SUMMER DIARRHEA — HEAT — HUMIDITY. LAWRENCE T. ROYSTER, M.D., NORFOLK 148

THE HYPODERMIC USE OF HEMATINICS IN THE TREATMENT OF ANEMIA IN CHILDREN. WITH REPORT OF CASES. H. LOWENBURG, A.M., M.D., PHILADELPHIA 160

PELLAGRA IN CHILDREN. A PRELIMINARY REPORT. J. ROSS SNYDER, M.D., BIRMINGHAM, ALA. 172

PROGRESS IN PEDIATRICS: THE WASSERMANN-NEISSER-BRUCK REACTION. PROF. DR. A. JESIONEK, GIESSEN. ABSTRACTED BY HARVEY PARKER TOWLE, M.D., BOSTON 180

INDEX OF CURRENT PEDIATRIC LITERATURE 189

OCTOBER, 1912. NUMBER 4

BLOOD-CULTURES DURING LIFE IN INFANTS AND YOUNG CHILDREN, WITH DESCRIPTION OF A NEW TECHNIC. MARTHA WOLLSTEIN, M.D., AND EDWARD MORGAN, M.B., NEW YORK 197

THE PANCREATIC FERMENTS IN INFANTS. ALFRED F. HESS, M.D., NEW YORK .. 205

INFANTILE ECZEMA AND INDIGESTION. PRELIMINARY CLINICAL STUDY WITH ILLUSTRATIVE CASES. HARVEY P. TOWLE, M.D., AND FRITZ B. TALBOT, M.D., BOSTON 219

INTESTINAL TOXEMIA IN THE NEW-BORN. JOHN LOVETT MORSE, A.M., M.D., BOSTON 229

STUDIES IN METABOLISM OF AMAUROTIC FAMILY IDIOCY. HENRY HEIMAN, M.D., SAMUEL BOOKMAN, PH.D., AND BURRILL B. CROHN, M.D., NEW YORK 234

PROGRESS IN PEDIATRICS: A SUMMARY OF THE RECENT LITERATURE ON ORTHOSTATIC ALBUMINURIA. MURRAY H. BASS, M.D., NEW YORK 246

CERTIFIED MILK IN INFANT FEEDING 256

INDEX OF CURRENT PEDIATRIC LITERATURE 258

NOVEMBER, 1912. NUMBER 5

THE INFLUENCE OF HIGH PROTEIN FEEDING ON THE GENERAL METABOLISM, ON THE INTESTINAL FLORA AND ON THE BODY TEMPERATURE OF INFANTS. L. EMMETT HOLT, M.D., AND P. A. LEVENE, M.D., NEW YORK 265

A BRIEF REPORT OF AN EPIDEMIC OF SORE THROAT WITH INVOLVEMENT OF THE CERVICAL LYMPH-NODES. JOHN RUHRÄH, M.D., BALTIMORE 301

EFFECTS OF COLD AIR ON BLOOD-PRESSURES OF CHILDREN AND YOUNG ADULTS IN VARIOUS STAGES OF TUBERCULOSIS. B. RAYMOND HOOBLER, A.M., M.D., NEW YORK 307

PAROXYSMAL HEMOGLOBINURIA. LOUIS C. AGER, M.D., BROOKLYN 311

ON THE MODE OF INFECTION IN EPIDEMIC CEREBROSPINAL MENINGITIS. DAVID BOVAIRD, JR., M.D., NEW YORK 317

INDEX OF CURRENT PEDIATRIC LITERATURE 323

DECEMBER, 1912. NUMBER 6

DIAGNOSIS OF ENLARGED BRONCHIAL GLANDS. HENRY FARNUM STOLL, M.D., HARTFORD, CONN. 333

A GRAPHIC CHART METHOD OF STUDYING AND TEACHING THE PRINCIPLES OF INFANT FEEDING. DAVID M. COWIE, M.D., ANN ARBOR, MICH. 360

HELMINTHIASIS IN CHILDREN. W. W. MURPHY, M.D., CHICAGO 378

SERUM TREATMENT OF PNEUMONIA. ROWLAND GODFREY FREEMAN, M.D., NEW YORK 383

LONG INTERVAL FEEDING OF PREMATURE INFANTS. J. C. LITZENBERG, M.D., MINNEAPOLIS 391

PROGRESS IN PEDIATRICS: RESPIRATORY SYSTEM. BORDEN S. VEEDER, M.D., ST. LOUIS 410

INDEX OF CURRENT PEDIATRIC LITERATURE 418

American Journal of Diseases of Children

VOL. 4 JULY, 1912 No. 1

LEUKOCYTIC "INCLUSION BODIES" WITH SPECIAL REFERENCE TO SCARLET FEVER*

JOHN A. KOLMER, M.D.
PHILADELPHIA

In November, 1911, Döhle[1] described certain "bodies" within the cytoplasm of the polymorphonuclear leukocytes in blood smears from thirty patients with scarlet fever. In a number of controls similar "bodies" were found in a patient with pneumonia and two patients with malignant neoplasms. As far as he knew these "bodies" had not been described before and to them the term "inclusion bodies" (Leukocytenenschlüsse) was applied. In March, 1912, Kretschmer[2] confirmed these findings and considered their presence in scarlet fever of diagnostic value. The latter also found these "bodies" in smears from patients with pneumonia and tuberculosis and two with diphtheria with streptococcus empyema. More recently Nicoll and Williams[3] reported a study of fifty-one cases of scarlet fever, finding the "bodies" in forty-five cases. Similar "bodies" were found in three out of twenty-five controls.

The presence of such "bodies" within the polymorphonuclear leukocytes of cases of scarlet fever are mainly of interest from the standpoint of differential diagnosis and of their possible relation to the etiology of this infection. Döhle suspected their relation to streptococcus infections and if these "bodies" are to be found in a large percentage of scarlet fever cases and are absent in non-scarlatinal conditions, they would be of considerable aid in differentiating true scarlet fever from cases of serum sickness with scarlatiniform rashes, rötheln, various toxic erythemata, etc.

Accordingly the object of this study was twofold:

1. To investigate the nature of the "bodies."

2. To determine their diagnostic value in scarlet fever after the examination of a large number of cases of scarlet fever, diphtheria, serum sickness and various infections, especially those of streptococcus origin.

*From the Laboratory of the Philadelphia Hospital for Contagious Diseases.
*Read before the Philadelphia Pediatric Society, June 11, 1912.

1. Döhle: Leukocytenenschlüsse bei Scharlach. Centraibl. f. Bakteriol. originale, 1911, lxi, part ½, p. 63.

2. Kretschmer, M.: Die Diagnostische Bewertung von Leukocyteneinschlüssen bei Scharlach. Berl. klin. Wchnschr., March, 1912, No. 11.

3. Nicoll, M., and Williams, A.: Inclusion Bodies in Scarlet Fever. Arch. Pediat., 1912, xxiv, No. 5, p. 350.

"INCLUSION BODIES"

These "bodies" are characteristically found in the protoplasm of the polymorphonuclear leukocytes of scarlet fever patients near the margin of the cell and are not connected with the nuclei. They present no fixed morphology but occur characteristically as rod and coccus forms. Due care must be exercised not to mistake the neutrophilic granules for round "inclusion bodies." Both forms may be present in any stage of scarlet fever and in the same smear. Early in scarlet fever most of the polynuclear cells will be found to contain one or more of the "bodies." The number of leukocytes showing their presence and the number of "bodies" per leukocyte vary according to the severity of the infection and diminish as convalescence is established. After the sixth day in mild scarlet fever they may require careful search before being found. The rod or bacillary type is most distinct in outline but all are more or less hazy in the best stained preparations.

TECHNIC

For examination, ordinary blood smears are prepared on perfectly clean slides. Thin smears are especially desirable. The "bodies" are readily stained by the Giemsa, Manson, Leishman, Wright and Jenner stains. Ordinary methylene-blue will bring them out. They do not stain with eosin and hematoxylin. In order to determine whether or not they are composed of chromatin, specific differential stains were employed to differentiate between chromatin and plastin. With the Giemsa stain the "bodies" are colored a faint bluish-green; with methyl-green and pyronin they present a fairly well-defined reddish color and with malachite-green and pyronin likewise a reddish color. They do not stain with iron hematoxylin. These stains would indicate conclusively that the "bodies" do not contain chromatin and are not protozoan in nature. They are probably composed of plastin, represent a degenerative process of the cytoplasm and are probably composed of spongioplasm. The fact that they are found in streptococcus infections would indicate their relation to the streptococcus. However, the systematic examination of the blood of eight rabbits receiving a single injection of streptococci from scarlet fever, puerperal sepsis and tonsils did not show the presence of these "bodies."

SCARLET FEVER

Blood smears from 216 patients with scarlet fever were examined, the majority being cases of mild or moderate severity. A few were of the severe anginose types of scarlet fever. Cases were examined from the first day of the rash to the fifty-second day after the rash had appeared. The results of examination of 200 cases are expressed in Table 1.

It will be noted that "inclusion bodies" are to be found in a large percentage of scarlet fever cases during the first few days of the infection. After the sixth day they become quite few in number and may be more difficult to find. Döhle did not find any after the sixth day but his series

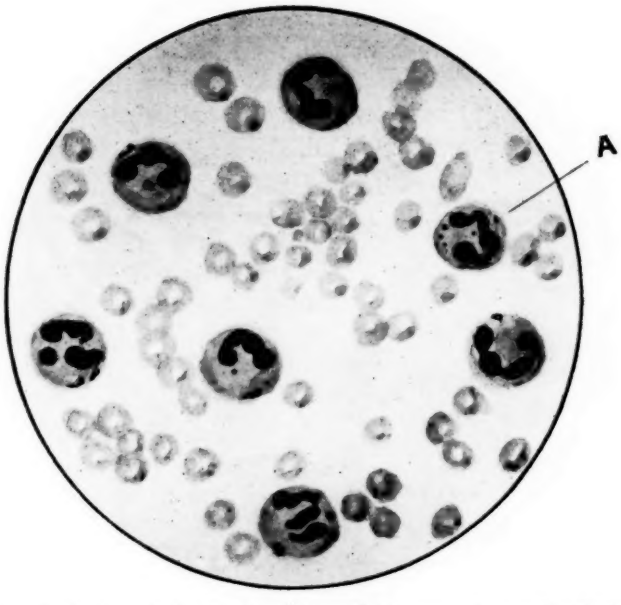


Fig. 1.—"Inclusion Bodies." Case of scarlet fever. A—neutrophilic granules.

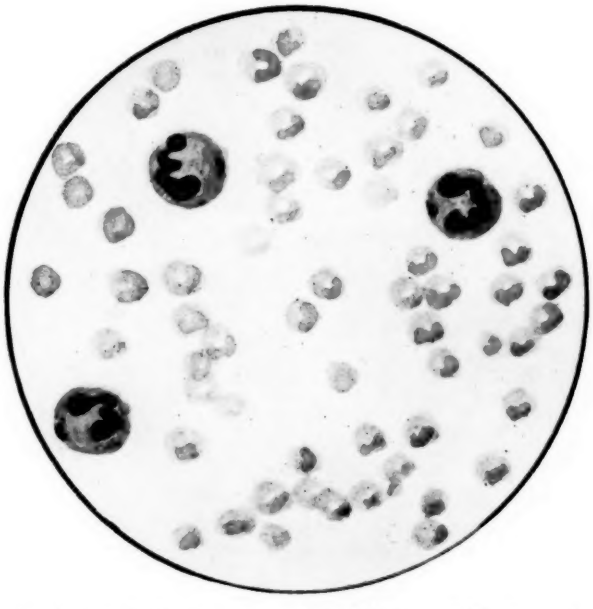


Fig. 2.—"Inclusion Bodies." Case of scarlet fever following extensive burns of the body.

was rather too small to be conclusive. I found one case, fully convalescent and about ready to be discharged from the hospital, showing typical "bodies" of irregular shape as late as the forty-fourth day. This series includes three cases of scarlet fever following extensive burns of the body, "inclusion bodies" being found in all.

TABLE 1.—RESULTS OF EXAMINATION FOR "INCLUSION BODIES" IN 200 SCARLET FEVER CASES

Day of Disease	Total Examined	Number Positive	Per cent. Positive
First	2	2	100
Second	14	14	100
Third	32	30	93.7
Fourth	14	12	85.7
Fifth	17	12	70.5
Sixth	15	10	66.6
Seventh	13	7	53.8
Eighth	12	3	25.0
Ninth*	9	0	0.0

*From the tenth to fifty-second days, seventy-two cases were examined with positive findings in six cases; one each on the tenth, thirteenth, seventeenth, thirty-sixth, thirty-eighth and forty-fourth days.

DIPHTHERIA

Smears from fifty patients with diphtheria were examined, mainly because the presence or absence of these "bodies" must be determined in this disease if such examinations are to be of value in differentiating serum sickness from scarlet fever. Not a few cases of diphtheria also show the presence of streptococci in the upper air passages along with the diphtheria bacillus.

Table 2 expresses the results.

TABLE 2.—RESULTS OF EXAMINATIONS FOR "INCLUSION BODIES" IN DIPHTHERIA

Day of Disease	Total Examined	Number Positive	Per cent. Positive
Second	4	1	25
Third	10	4	40
Fourth	8	5	62.5
Fifth	8	0	..
Sixth	4	0*	..

*From the sixth to the twenty-first day, 16 were examined, with three positive, all occurring on the seventh day.

It will be noted that these "inclusion bodies" are to be found in a high percentage of diphtheria cases during the first four days of the infection. Therefore if a serum rash should develop within this period the presence or absence of "inclusion bodies" could have no diagnostic importance in differentiating the rash from that of true scarlet fever.

SERUM SICKNESS

Of all the conditions resembling scarlet fever, serum sickness presenting a scarlatiniform rash is most difficult of diagnosis. This is especially true in a hospital for contagious diseases where one must be constantly

on guard to detect scarlet fever and where it is almost impossible to prevent an occasional outbreak in the diphtheria wards. Fortunately most serum rashes are urticarial in nature and easily diagnosed. Scarlatiniform rashes are also prone to develop from the third or fourth to the tenth day after receiving serum and this is a source of further confusion because this period coincides so closely with the usual incubation period of scarlet fever. In my experience the proper diagnosis and management of such cases becomes one of the greatest problems in the hospital management of contagious diseases.

Of thirty cases of serum sickness presenting urticarial rashes, developing, on the average, ten days after admission to the hospital, none showed the presence of "inclusion bodies." Practically all of these cases were examined within twenty-four hours after the appearance of the rash.

I had the opportunity of studying fourteen cases presenting scarlatiniform rashes, thirteen occurring in the diphtheria wards, with the following results:

1. Five were diagnosed clinically as serum sickness and none showed the presence of the "bodies."

2. Five were diagnosed as true cases of scarlet fever and all showed the presence of the "bodies."

3. Two were diagnosed as true scarlet fever but did not show the presence of the "bodies." Both of these cases were very mild in character and developed on the twelfth and thirty-second days, respectively, after admission to the diphtheria wards.

4. One case showed the presence of "bodies" but was of doubtful clinical diagnosis. The child may have had a rash before admission to the hospital but the throat was very much injected and resembled the angina of scarlet fever. The cervical glands were likewise enlarged.

5. Case 14 was that of Dr. C. P. Brown who presented a true scarlatiniform rash over the body but with no other symptoms of scarlet fever. This rash was of gastro-intestinal origin. The leukocytes did not show the presence of "bodies."

MEASLES, ERYSIPELAS, PUERPERAL SEPSIS, ETC.

The blood of seventy-six patients suffering with various infections other than scarlet fever, diphtheria and serum sickness was examined. Most interest centers about those which may be mistaken clinically for scarlet fever and also about those infections due to streptococci.

(a) Measles.—Twelve cases, varying from the third to the sixth day after the appearance of the rash, were examined and all were negative. One case was complicated by bronchopneumonia.

(b) Rötheln.—But one case was examined, on the second day after the rash made its appearance, and this was negative.

(c) Erysipelas.—Eleven cases were examined with positive findings in seven. Of these seven positive cases inclusion "bodies" were found on the third, fourth, fifth, ninth, tenth, twenty-fourth, and twenty-fifth days after the onset of symptoms.

(d) Puerperal Sepsis.—One case due to a *Staphylococcus aureus* infection was negative; a second case, infection not determined, showed typical "inclusion bodies."

(e) Pneumonia.—Three cases of croupous or lobar pneumonia were examined and in two of these, "bodies" were found. Two cases of bronchopneumonia were negative. Of two cases of empyema, not included in these pneumonia cases, one was positive, and in this case streptococci were found in the pus.

In none of the forty-three additional cases including varicella, pelvic inflammatory disease, pericarditis, goiter, furunculosis, eczema, gonorrhea, mastoiditis, gastro-enteritis, chronic lymphatic and splenomyelogenous leukemia, pernicious anemia, etc., were "inclusion bodies" found.

The presence of these bodies in such a large percentage of cases of erysipelas, in the case of streptococcus empyema and in one of two cases of puerperal sepsis which was likely of streptococcus origin, indicates strongly the relation between these "inclusion bodies" and streptococcus infections.

CONCLUSIONS

1. "Inclusion bodies" are composed of a plastin, probably spongioplastin, and are related to the presence of streptococci.

2. They are present in the polymorphonuclear leukocytes of 94 per cent. of scarlet fever cases during the first three days after the onset of the disease. After this they diminish in frequency and are generally absent after the ninth day.

3. They are to be found in 42 per cent. of diphtheria cases during the first three days of the disease; after this time they are but seldom found.

4. "Inclusion bodies" are found not only in scarlet fever, but in other streptococcus infections.

5. The diagnostic value of these "bodies" is necessarily limited. In serum sickness with a scarlatiniform rash their absence excludes scarlet fever with a fair degree of accuracy. Their presence in this condition, however, may not be due to scarlet fever but to the primary attack of diphtheria. They have, therefore, a negative value.

6. An examination of the blood for these "bodies," however, is very simple and possesses value in aiding a differential diagnosis between scarlet fever, rötheln, measles and gastro-intestinal rashes.

I wish to express my thanks to Dr. A. J. Smith, Dr. H. K. Meyers, Dr. S. S. Woody, Dr. E. Laubaugh and Dr. G. Faries for aid in the work.

Second and Luzerne Streets.

THE CLINICAL SIGNIFICANCE OF ABNORMAL RESPIRATION AND DEGLUTITION CLICK OR FREMITUS; THE PNEUMONIC-RESPIRATION-PAUSE-CYCLE

LAMBERT OTT, M.D.
PHILADELPHIA

As respirations normally differ at the different periods of life, so abnormal respiration has a parallel difference as a manifestation of disease in the different periods of life. I may cite, for example, the puerile lung sounds in childhood and adolescence and the diminished and distant respiratory sounds of old age; also the rapid respirations in infantile life and the slower respirations of advanced age; the brief post-expiratory pause in early life and the longer post-expiratory pause in advanced age.

The respirations of exhaustion are in a manner influenced mostly by the age of the patient and partly by the degree of exhaustion.

The breathing of the ill baby or child in exhaustion is shallow, rapid and pauseless, indicating gravity and often impending death. If they fall into slumber while in this condition the respirations lessen in frequency, lengthen in range and are still pauseless. If they awaken unimproved the breathing resumes the characteristics just described; but if, on the contrary, there is an improvement in the patient's condition, the respirations are less rapid, range lengthened, calmer, and an occasional normal post-expiratory pause is noticeable. When in the above clinical picture the disease had merely a remission and a relapse occurs, there is manifested at once a return of rapid and shallow respiration, with no inter-respiratory pause; sometimes this phenomenon precedes the increase of temperature, a natural and usual accompaniment of a recrudescence in disease. When the degree of exhaustion increases, presaging death, respiration becomes a little arhythmical, the inspiratory range being lessened and expiration being more prolonged, this irregularity becoming more accentuated in ratio as the end approaches. The final expiration is well prolonged, with an occasional pause, while a second feeble and aborted inspiration follows, ending in more of an expiratory spasm in which the muscles other than those directly concerned in breathing become involved.

Respirations of middle life in exhaustion differ somewhat from those in earlier life. They are more rhythmical than in infantile life, shallow and rapid, and with no inter-respiratory pause. The ranges of inspiration and expiration are longer and as death approaches the occasional post-expiratory pause is noticeable. The respirations in exhaustion of

advanced age present some peculiarities. Inspiration and expiration follow each other without pause, have a longer range than in earlier life, are irregularly rhythmical, sometimes expiration being longer than inspiration, but soon again rhythm is assumed, and then a rhythm favoring longer expiration. As the exhaustion increases and the patient grows weaker, respirations grow more shallow and a post-expiratory pause is established and prolonged, which continues until death. The latter manifestation is in striking contrast to the post-expiratory pause in infantile exhaustion which is only occasionally apparent and not as continuous as in the last moments of the aged.

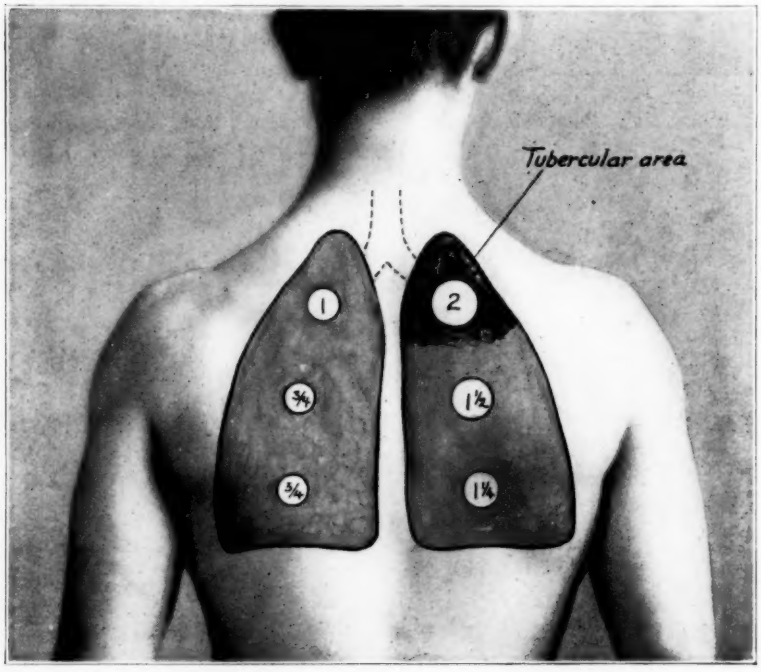


Figure showing by circles posteriorly the different degrees of deglutition fremitus or click in apical tuberculous infiltration of the right lung as compared with the healthy lung.

Respiration of pneumonia in infancy and childhood has a positive and pathognomonic post-inspiratory pause. This pause is not continuous as many suppose. There are usually three to five post-inspiratory pauses, then follows rapid rhythmical respiration without a pause, which ends in one post-expiratory pause and then the post-inspiratory pauses begin again. There is a definite cycle in the recurring post-inspiratory pauses which vary in number from two as a minimum and six as a maximum before the period of rapid pauseless respiration sets in. But the peculiar

point observed is that just at the ending of one phenomenon and before the beginning of the recurrence of the period of the post-inspiratory pause, one prolonged expiratory pause occurs, seeming to link the two changes in some unexplainable physiologic manner. In other words, between the pauseless respirations succeeding the post-inspiratory pauses there appears one prolonged post-expiratory pause, thus leading to the beginning of the cycle. I might name this symptom the pneumonic-respiratory-pause-cycle. It must be remembered that this peculiar respiratory-pause-cycle is generally found in infantile and childhood pneumonia. Infants under 18 months or children 2 years old manifest this condition more strikingly than older children. But let this same pneumonic child fall into sound or natural sleep and all arhythmia of respiration ceases; then you have still continuing the rapid respirations, but slower than when awake, and absolutely rhythmical and pauseless in occurrence. On reawakening the respiratory-pause-cycle is resumed. Whenever the post-inspiratory pause began to lessen and respiration grew less rapid the pause gradually became more post-expiratory and lessened post-inspiratory. In other words, a transposition of the pause from post-inspiration to post-expiration was a more favorable sign; the lessening of the respiratory pause or its absolute absence coupled with increased rapidity of respiration indicated a tendency to exhaustion and gravity.

I recall a case of bronchopneumonia in an infant 12 months of age with cyanosis, bradycardia and no cardiac murmurs, all indicating a weak heart and the overwhelming severity of the attack. The respirations differed somewhat from the regular type in that inspiration was shorter than expiration. There was the post-inspiratory pause and then a prolonged expiration, pauseless but wheezy like the expiratory wheezing of an attack of bronchial asthma in the adult. Otherwise, the pneumonic-respiratory-pause-cycle was manifested throughout the disease until some hours before death, when rapid and pauseless respiration followed and continued to the end.

Later observations convince me that the pneumonic-respiratory-pause-cycle is nearly always a manifestation of bronchopneumonia, the pneumonia of infantile life, and found in croupous pneumonia when occurring in a child 2 years of age and under. Pneumonia in older children presents less of the respiratory pause-cycle, but one finds a more continued and more pauseless respiration and the altered pulse-respiration ratio is more marked than in earlier life. I have sometimes found a pleuritic complication attended with pain in inspiration productive of irregular pauses in respiration, evidently a suspension of breathing to alleviate pain.

My experience leads me to believe that in cases of children of lowered vitality from disease other than lung troubles, manifesting cerebral symptoms, regularity of respirations negatives organic brain disease. This rule can also be applied to the pulse.

If an infant having apical pneumonia with the characteristic respirations develops cephalic symptoms, and the breathing does not change from the pneumonic-respiratory-pause-cycle, one may conclude that the brain symptoms are merely symptomatic and not organic in character. The absence of persistent vomiting is confirmatory of this conclusion.

Parenthetically, I have observed another point while closely studying the respirations of pneumonic children:

If one places the hand on the right and left flanks of the sick child one will notice a greater sinking of the side having the pneumonic lung. This observation I have frequently verified.

In the respiration of pneumonia of adults the normal respiratory pause is entirely missing, inspiration and expiration following each other in close succession. As exhaustion increases respiration grows more rapid, shallow and somewhat labored. The moment the crisis begins there is a return of an occasional post-expiratory pause and less rapid breathing.

In the early convalescence of adult pneumonia during sleep respiration becomes slower and the normal post-expiratory pause is continuous; even when during the preceding waking there is not a continuous post-expiratory pause, with some accelerated pauseless breathing.

In a case of adult pneumonia from which the above observations were made I noticed the following: One-half to one minute before awaking respirations became pauseless and rapid and remained so until semi-consciousness merged into full consciousness when approximately normal breathing followed. This manifestation is probably more physiologic than pathologic.

In the extremely aged there is a form of respiration accompanying bronchial catarrh in which there is shallow and accelerated breathing, with only an intermitting post-expiratory pause, with moist and dry râles, indicating presumably a pulmonary edema, the other symptoms not being in accord. I ascribed this condition to an associated nervous element and it is seemingly more dangerous than real. The same clinical picture in a young adult would portend greater evil.

By the term deglutition fremitus I mean the sounds occasioned by the act of swallowing which are more readily conveyed to the ear through the area of a pneumonic induration. In the adult it does not seem as valuable as the transmitted voice, but in infantile pneumonia, where the act of crying is the sole manner of expression, this diffuses itself throughout the lungs in such a way as to confuse. Let the suckling infant nurse and while the ear is auscultating the lungs posteriorly note the area conveying the noise of the act of swallowing. A gurgling or clicking sound heard more distinctly at any point will indicate a pneumonic spot. Repeatedly I have verified this observation but it requires tact, time and patience to derive the benefit from this aid to the diagnosis of pneumonia.

While other well-known symptoms are more positive diagnostically, a decided aid in localizing indurated areas may be obtained by deglutition fremitus. Could we but request the infant to modulate the sound of crying or soften the voice we would have an equally valuable aid in the diagnosis of lung consolidation as in adult vocal fremitus, but this is impossible.

The examination should be made after the infant has fasted for a long time and then when put to the breast it nurses with a feverish thirst and hunger and consequently swallows oftener and greater quantities, which aids in accentuating the deglutition fremitus through indurated area.

I am justified in calling deglutition fremitus, deglutition click, the pronunciation of the latter word "click" corresponding to the fremescent sound heard when auscultating the chest during the act of swallowing. The following peculiarity developed in the course of my examination: Over a left apical tuberculous indurated area posteriorly the deglutition click or fremitus was decidedly distinct, and as I passed down to the middle of the lung over a non-indurated area the fremitus was slightly lessened. Over the base of the apically indurated lung, or over the normal lung tissue, the fremitus was still accentuated but less distinct than that of the middle area and the latter fremitus was less distinct than that of the apical area.

Examining the healthy lung of the same individual, deglutition fremitus was alike in pitch and tone over the entire lung, possibly a little more accentuated at the apex. But the three distinct degrees of deglutition fremitus of the healthy lung were measurably less than over any area of the apically diseased lung.

In the accompanying illustration I can better explain the difference in intensity of sounds as evidenced over the lung area of one lung partly diseased and the lessened deglutition fremitus of its non-diseased fellow.

Let an imaginary circle 2 inches in diameter be drawn over the indurated lung area posteriorly and one circle 1½ inches in diameter over the middle non-indurated lung area of the same lung, and a third circle 1¼ inches in diameter over the base of the same lung. Then draw a circle of 1 inch over the apex posteriorly to the opposite healthy lung, and two circles of three-fourths inch in diameter over the middle and basilar area of the same lung. You will now have a guide as to the variety of fremitus produced by the act of deglutition. The 2-inch circle represents the maximum of intensity over the tuberculous, infiltrated apex and the three-fourths-inch circle represents the base of the opposite healthy lung — the most remote area from the diseased tissue — giving the less distinct deglutition click or fremitus; the three-fourths-inch circle

representing a minimum as compared with the 2-inch circle over the tuberculous area as a maximum of fremitus.

My studies and observations in deglutition fremitus or deglutition click are yet embryonic and this paper merely embodies a preliminary report. I do not believe the developments thus far contribute much in the way of a decided and valuable aid to diagnosis, but my purpose is to call the attention of the profession to a hitherto neglected element in physical diagnosis which can be further evolved on a practical basis by the great body of medical practitioners, who are the final arbiters of all the novelties in medicine and diagnosis. They must affirm or reject a proposition on its merits. The child cannot be asked to say one, two and three, or be requested to alter its respiration like an adult, but the act of swallowing is instinctive and automatic and can be readily produced even in a new-born infant.

831 North Broad Street.

THE RELATIONSHIP BETWEEN THE INFECTION IN THE CHILD AND CLINICAL TUBERCULOSIS IN THE ADULT *

FRANCIS MARION POTTENGER, A.M., M.D., LL.D.
MONROVIA, CAL.

Most painstaking inquiry into the clinical history of patients suffering from clinical tuberculosis fails, in nearly all instances, to reveal with any degree of certainty the time when the infection occurred. Especially is this true in adults, for tuberculosis is not the disease that we have long thought it to be. The time of infection and the time when clinical symptoms manifest themselves is, as a rule, most probably separated by years. We must not expect an exposure to infection and an immediate manifestation of symptoms. It is just as important, in fact, more important, to know that the patient associated intimately with some one suffering from tuberculosis when a child, as to find an exposure immediately prior to the manifestation of symptoms. Some German authors,[1, 2, 3] are considering tuberculosis as a disease of three stages, the same as syphilis; first, that of the infection of the regional lymph-nodes; second, the stage when the disease spreads to new areas, and third, the late manifestations.

The cutaneous tuberculin test as given us by von Pirquet has done more to help us understand the nature of tuberculosis than any other means.

Hamburger[4] says he agrees with those observers who believe that if a child develops tuberculosis within the first two years of life a careful examination into the clinical history will determine the source of infection in the majority of cases. This can well be done during the first two years of life, for during this period tuberculous infection is comparatively rare, and when it does come it usually manifests itself in one of the rapid

*Read in the Section on Diseases of Children of the American Medical Association at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

1. Ranke: Vortrag in der Gesellschaft für Kinderheilkunde. München. med. Wchnschr. No. 15, April 12, 1910, p. 826; Ueber den zyklischen Verlauf der menschlichen Tuberculose, Brauer's Beitr. z. Tuberkulose, 1911, xxi, Part 1.

2. Von Pirquet: Vortrag von dem Deutschen Zentral Committee zur Bekämpfung der Tuberkulose, Berlin, June, 1911. Cited by Ranke.

3. Hamburger: Ueber Spätformen der Tuberkulose. München. med. Wchnschr., March 19, 1912.

4. Hamburger: Allgemeine, Pathologie u. Diagnostik des Kindertuberkulose, 1910. Die Tuberkulose als Kinderkrankheit. München. med. Wchnschr., No. 25, 1908, No. 25.

forms, developing soon after the inoculation and showing threatening symptoms as quickly as the disease can develop; and usually proving rapidly fatal. There is almost no tendency to chronicity in the disease as it manifests itself in these very young children. As the child becomes older, however, the disease assumes more of a chronic course, which is another way of saying that the child has begun to manifest a degree of immunity toward the tubercle bacillus.

This fact is easily explained according to the modern theories of immunity and the explanation is borne out by experimental evidence. Individuals suffering from tuberculosis are found everywhere and many of them do not know that they have the disease until long after it becomes an open lesion. The consequence of this is that bacillus-bearing sputum is scattered carelessly here and there, wherever human beings dwell. Tuberculous cattle are also found in nearly all of the dairy herds, thus infecting quite a large amount of the milk which is consumed. The child begins his first fight with the bacillus, then, as soon as he begins to come in contact with people and take food produced otherwise than by his mother's breast; and particularly when he reaches the age of playing on the floor, for it is a well-established fact that tubercle bacilli are thickest on the floor and near the floor. They are carried into the home in one way or another and remain viable for long periods of time. This is also the time when the child begins to eat food, the production and preparation of which are difficult to control, and the time when the child begins to associate with other children.

The question of infection is a question of dosage of bacilli. If the child takes in only a few bacilli at first, they stimulate the child's cells, antibodies are produced and the invading bacilli are destroyed with a resultant increase in the child's antibodies, or, in other words, a heightened immunity or resistance is established against further infection with tubercle bacilli. The child is now fortified against a larger dose of bacilli than before. This same thing, inoculation followed by destruction of the bacilli, is repeated time after time until quite a degree of immunity is gradually established; but not enough, however, in most instances, to protect the child from an infection occurring, as is shown by the fact that nearly all children are infected with tubercle bacilli before the fifteenth year is reached, according to the statistics of Hamburger, von Pirquet, Gangenhofer and others. Nearly every child comes in contact with more bacilli than he can overcome sometime before he reaches the age of puberty. That all of these children do not develop clinical tuberculosis is well known; so the inquiry "What becomes of these infections?" is pertinent. A small percentage develop acute tuberculosis and die quickly; another comparatively small percentage develop a chronic tuberculosis; another group shows no definitely recognizable symptoms that

have been heretofore considered as due to tuberculosis, but suffers from the infection and fails to develop a natural physical strength; while the great majority remain free from symptoms and develop in a normal manner, as far as we are able to tell. The chances of the child showing acute symptoms diminishes with age, there being comparatively less acute tuberculosis after the fourth year, as shown in Tables 1, 2 and 3.

The after-history of these last two classes shows the development of quite a large percentage of our clinical tuberculosis; and it is the explanation of the clinical disease and its relation to the early infection that I now wish to consider.

CLINICAL TUBERCULOSIS AND EARLY INFECTION

The evidence adduced from animal experiment is very important and convincing. The presence of a tuberculous focus, either healed or unhealed, affords a certain though variable degree of immunity to the animal harboring it. The writer elaborated this point quite fully in a paper read before the Medical Society of Greater New York, March, 1910,[5] and will quote from it.

"For some time it has appeared to me that patients suffering from tuberculosis must develop a certain degree of immunity to the bacilli producing their own infection at least; otherwise they would have no chance for life against the countless myriads of bacilli which are thrown into their air passages and which are swallowed by them daily during the period of a long illness. But in spite of this opportunity for inoculation, which is present even for years in many instances, patients recover. If the number of bacilli which are found more or less constantly in the air passages of patients with open tuberculosis of a moderately advanced degree were placed in the air passages of healthy individuals, or, if the number swallowed by such patients were injected into the alimentary canals of healthy individuals, we certainly would expect infection to occur.

"Recent experiments of Calmette,[6] Hamburger,[7] Römer[8] and others show that through the first infection an immunity is developed, which affords protection against further infection from both the bacilli within the body and from those coming from outside sources. Guinea-pigs, rabbits, sheep and cattle which have been once infected with tuberculosis show a resistance to a second infection that can only be accounted for by the presence of a considerable degree of immunity.

"It is slf-evident that the greater the degree of immunity present the more chronic the disease. This is noted in the manner in which tuberculosis destroys people who have not been previously inoculated with the disease, such as the Africans and people of the Faroe Islands, as compared with the way it affects the inhabitants of Europe and America. It is also noted in children in the early months of life when compared with children in later years. During the first year

5. Pottenger: Immunity in Tuberculosis Considered from Both the Experimental and Clinical Standpoints. Med. Rec., New York, June 18, 1910.

6. Calmette: Nouvelle contribution à la étude de la Vaccination des bovides contre la Tuberculose. Ann. de l'Inst. Pasteur. September, 1908.

7. Hamburger: Die pathologische Bedeutung der Tuberkulin Reaction. Wien. klin. Wchnschr., 1908, No. 29.

8. Römer: Weitere Versuche über Immunität gegen Tuberkulose durch Tuberkulose zugleich ein Beitrag zur Phthiseogenese. Beitr. z. Klin. der Tuberkulose, 1909, xi, and xiii.

of life tuberculosis is nearly always acute, assuming the meningeal or acute miliary form. Hamburger[2] gives the following interesting statistics of tuberculosis in childhood as presented by post mortem evidence at the St. Ann Kinderspital, Vienna, from 1903 to 1906, inclusive.

TABLE 1.—MORTALITY OF TUBERCULOSIS BY AGE PERIODS

Periods	From Birth to 3 mos.	4-6 mos.	7-12 mos.	Total	2 years	3-4 years	5-6 years	7-10 years	11-14 years
Number of cases...	4	13	32	49	74	102	38	41	31
Fatal cases	4	13	27	44	51	69	23	28	16
Per cent. of fatality	100	100	80	90	70	67	60	68	50

TABLE 2.—FREQUENCY OF HEALED TUBERCULOSIS BY AGE PERIODS

	1 year	2 years	3-4 years	5-6 years	7-10 years	11-14 years
Total number of cases	49	74	102	38	41	31
Number with signs of healing..	0	0	7	4	7	10
Per cent. of healed tuberculosis .	0	0	7	10	17	33

"He also cites the following table (Table 3) from Hamburger and Sulka:

TABLE 3.—RELATIVE FREQUENCY OF ACUTE AND CHRONIC TUBERCULOSIS BY AGE PERIODS

	1 year	2 years	3-4 years	5-6 years	7-10 years	11-14 years
Miliary or meningeal tuberculosis	13	19	28	10	8	2
Chronic general or pulmonary tuberculosis.....	7	6	6	1	4	6
Total fatal cases	20	25	34	11	12	8

"These statistics show that tuberculosis is nearly always fatal in the first years of life; in 90 per cent. the first year, 70 per cent. the second, with a gradual decrease so that by the time puberty is reached the mortality has fallen to 50 per cent. Another interesting fact is found in Table 2. No tendency to healing is manifested during the first and second years and only in 7 per cent. in the third year. By the time puberty is reached signs of healing appear in 33 per cent. Table 3 shows that of twenty cases in the first year of life, thirteen were cases of acute miliary or meningeal tuberculosis; of twenty-five cases in the second year nineteen were acute miliary or meningeal tuberculosis, and of thirty-four in the third year, twenty-eight were of this form, while between the eleventh and fourteenth years of eight cases only two were of these acute forms."

The facts adduced by these clinical observations and experimental results warrant the assertion that an individual who has a tuberculous lesion already present in the body develops a marked protection against further inoculation with bacilli, and that if a further extension of the disease is to occur from the focus already within the body, or a new infection is to occur from without, it must be by an inoculation of sufficient bacilli to overcome the increased protection which is present as a result of the previous infection. For conclusive proof of this point I would refer to the experiments detailed by Römer in his most valuable report.[6]

That such an inoculation occurs now and then in adult life we can readily believe, especially in such instances as where a husband develops the disease after a wife, or vice versa, when sufficient precautions have not been taken; or when a brother or sister follows another brother or sister. Even in these cases, however, we are not sure that the infection comes from without. In many such cases that I have examined I have felt that it was just as probable that the disease in the second member was from an old focus from within and that the cause of the lesion was an extension resulting from the lowered resistance caused by the reduced physical and depressed mental condition of the patient.

The usual clinical history of patients suffering from tuberculosis when carefully analyzed leaves little doubt that the disease as it presents itself is either a new activity in, or an extension from, an old focus. In those cases in which the first symptom is hemoptysis, or those coming on, following some illness such as influenza, bronchitis or pneumonia, or those in which it comes on suddenly with cough, fever and expectoration, there is little doubt that the acute symptoms are a manifestation of activity in an old focus. In those cases in which the illness comes on slowly, with feelings of indisposition, slight digestive disturbances, a gradually developing cough, the question is a little more difficult to decide; but careful consideration will place most of these in the same class. It is impossible to obtain a history in more than a minor portion of our tuberculous cases wherein we could feel at all certain that the given infection was due to a definite exposure to bacilli.

PREVENTION

The preponderance of clinical evidence, then, throws our clinical tuberculosis, as it manifests itself in adult life, back to those early infections which occur in childhood. It is essential to understand this relationship because of its bearing on the rational measures to be adopted for its prevention and cure.

It is these early infections which occur in childhood that eventually cause the clinical tuberculosis in adults, and which are responsible for the enormous mortality from this disease; then, the stamping out of tuberculosis, if it is ever attained, must come through either preventing the

infection from occurring, holding it in a state of quiescence, or healing it out when it has once occurred. It seems almost an impossibility to think of being able to control the large majority of cases of open tuberculosis, which would be necessary in order to stamp out the disease by taking away the danger of infection. This difficulty is greatly increased by the fact that the open stage is often reached months and even years before the diagnosis is made. While we must surround the open cases with the most approved hygienic measures so as to take away every possible source of infection, yet I cannot believe that such measures alone will ever stamp out tuberculosis. It must be stamped out by making the individual immune to infection or by curing the infection when it has occurred, thus preventing it from reaching the open stage.

Personally, I believe that the prevention of tuberculosis will come through vaccination; and, if I interpret the works of our leaders rightly, such a desideratum is not as far off as many believe. The experimental studies on immunity made by such men as Koch, Behring, Maragliano, Wright, Römer, Wolff-Eisner, Calmette, Spengler, Trudeau, von Ruck, and others, and the very interesting reports by the use of the living bacillus, made by Webb, are blazing the way for one of the greatest boons to the human race, the production of a positive immunity.

We, as physicians, however, cannot wait for this to come about, but we must continue attacking the problem in every way that offers a partial solution. We must continue our educational measures, especially instructing those afflicted with the disease in the methods of preventing its spread. Remembering the great percentage of the human family that harbors foci of infection, we must push the campaign for more hygienic living, thus hoping to prevent quiescent lesions from taking on activity. We must endeavor to cure as many patients suffering from clinical symptoms as possible.

But, what of these children who are infected? Should we disregard them as we have in the past and wait for them to become clinically tuberculous before offering them help? Of course we realize that while it is possible that at least 75 per cent. of all children are infected before they reach the fifteenth year, probably not more than 20 per cent. will develop what we are able to diagnose as clinical tuberculosis, and not more than 10 or 15 per cent. will die of the disease. This means that the children have three chances out of four of never knowing that they have an infection if left alone, and that they have about six chances out of seven of not dying of the disease. The question for us to settle is this: Is the percentage of morbidity and mortality, when compared with the total number of infections, sufficient to warrant the medical profession in attempting to heal out all the infections which can be determined in childhood? The recognized rule in syphilis is to heal out all those who show

a positive Wassermann. The best thought in medicine and surgery is to make early diagnoses and apply measures for the eradication of disease as early as possible; and, as a result, deaths from such diseases as diphtheria, appendicitis and gall-bladder infections are becoming rare. Shall we treat tuberculosis the same? If so, then it is the province of the pediatrist to control clinical tuberculosis. We have the various tuberculin tests at our command. They can be given with ease and without fear of harm. They are specific in their action. If the operator will learn the few conditions under which they may sometimes fail to react, even though an infection be present, he will have at his command a most reliable diagnostic method. Conditions which cause the tuberculin reactions to remain in abeyance, even though infection be present, are as follows: an advanced tuberculosis, a cachectic condition, a very low state of nutrition and the presence of some of the acute infectious diseases, notably measles. When these conditions are present a negative reaction does not signify that tuberculosis is not present.

If it is right to treat every patient for syphilis if he reacts to the Wassermann test, is it not equally just to afford the patient reacting to tuberculin the same protection? Is not the comparative greater seriousness of the disease an argument for such a course?

I believe that it is the physician's duty to seek out these children who are infected and put them on a hygienic regimen which will favor a natural physical development. Those in whom the disease shows signs of activity should be treated with tuberculin as well as with the usual hygienic dietetic measures; for we now have sufficient clinical data to show beyond doubt that a greater percentage of patients treated with hygienic and dietetic measures, plus tuberculin, heal out than where the disease is treated with hygienic measures alone. It might further be stated as an additional reason for treating infected children that they respond better to treatment than adults do.

RECENT ADVANCES IN OUR KNOWLEDGE OF MEASLES

JOHN F. ANDERSON
Director Hygienic Laboratory
AND
JOSEPH GOLDBERGER
Passed Assistant Surgeon, U. S. Public Health and Marine-Hospital Service
WASHINGTON, D. C.

While it has been quite the general belief for years that the infection of measles is contained in the blood and in the nasal and buccal secretions and, perhaps, in the "scales," it is surprising on what inconclusive data this belief has been based.

Previous to 1905 the data as to the infectivity of the blood, while highly suggestive, were by no means conclusive; but in that year Hektoen reported the infection of two men with measles by inoculation with an ascitic broth culture of blood drawn from human cases of the disease during the first thirty hours of the eruption. These results left no doubt as to the presence of the virus of measles in the blood during at least the first thirty hours of the eruptive period.

The state of our knowledge as to the presence of the virus in the nasal and buccal secretions was even more unsatisfactory than that in regard to the blood, although clinical observations and Mayr's experiments on children had made it fairly certain that these secretions were infective for man, and the same author's negative experiments with the "scales" had also shown that it was fairly certain that these did not contain the virus of the disease.

The literature of measles contains many reports of attempts to produce measles by various procedures in animals other than monkeys, but Josias[1] seems to have been the first to attempt to inoculate the monkey with measles. His work had been inspired by Chavigny's[2] reported observation of a monkey that had developed measles after having been in close contact with a case of the disease in the person of its keeper. In 1898 Josias reported attempts to infect monkeys with measles by various methods and he apparently obtained positive results in some instances.

---

1. Josias, Albert: Recherches expérimentale sur la transmissibilité de la rougeole aux animaux. Med. moderne, Paris, 1898, ix, p. 153.

Mayr, Frantz: Beobachtungen über Masern, etc. Zeitschr. d. k. k. Gesellsch. d. Aerzte zu Wien, 1852, i, 13; Masern. Virchow's Handb. der special Path. u. Therap., 1860, iii, Part 3, p. 106.

2. Chavigny, Paul: Un cas de rougeole chez un singe. Bull. méd. Paris, 1898, xii, p. 334.

Grunbaum[3] in 1904 reported attempts to infect two chimpanzees by various means, including the injection of blood drawn from the median basilic vein of patients with measles, but he regarded his results as negative.

On account of the prominent place occupied by measles in our morbidity and mortality statistics, it appeared to us highly important that studies be made on the disease to determine its cause, means of transmission and prevention. In view of the apparent insusceptibility of the lower animals to measles it seemed to us most desirable that the susceptibility of the monkey to the disease be thoroughly tried out.

AUTHORS' EXPERIMENTAL WORK

In pursuance of this plan we began our work[4] on experimental measles early in the summer of 1910 by inoculating two rhesus monkeys with blood from a patient with measles. In each of the animals a slight rise in temperature, eleven days after inoculation, was noted, but an interpretation of its significance was not possible.

With blood from our second patient we also inoculated two monkeys with a result similar to our first case. With blood from our third patient we inoculated three monkeys. Ten days after inoculation there was noted a slight rise in the temperature of one of the animals. At the same time there appeared a few papules on the face, brows and chin with a diffuse erythema of the brows and eyelids. The rise in temperature was not sustained. At the end of four days the eruption had perceptibly faded, and a fine branny scaling was noted at the site of the fading papules. This result we were inclined to consider as indicating a mild attack of measles, even though an attempt at passage with blood drawn soon after the eruption was first noted, resulted negatively.

The thought was constantly present in our minds from the beginning of our work with measles that we should obtain blood for inoculation as early in the disease as possible, and fortunately we saw our fourth patient at not less than fourteen hours after the eruption first appeared. With blood drawn from this patient about fourteen hours after the eruption was

3. Grunbaum, A. S.: Some experiments on enterica, scarlet fever, and measles in the chimpanzee; a preliminary communication. Brit. Med. Jour., April 9, 1904, p. 817.

4. Anderson, John F., and Goldberger, Joseph: Experimental Measles in the Monkey: A Preliminary Note. Pub. Health Rep., June 9, 1911, p. 847; Experimental Measles in the Monkey: A Supplemental Note. Pub. Health Rep., June 16, 1911; The Period of Infectivity of the Blood in Measles. Jour. Am. Med. Assn., July 8, 1911, p. 113; Goldberger, Joseph, and Anderson, John F.: An Experimental Demonstration of the Presence of the Virus of Measles in the Mixed Buccal and Nasal Secretions. Jour. Am. Med. Assn., August 5, 1911, p. 476; The Nature of the Virus of Measles. Jour. Am. Med. Assn., Sept. 16, 1911, p. 972; Anderson, John F., and Goldberger, Joseph: The Infectivity of the Secretions and the Desquamating Scales of Measles. Jour. Am. Med. Assn., Nov. 11, 1911, p. 1612.

first noted, and before the temperature had reached its maximum, we inoculated two rhesus monkeys. Ten days after inoculation the temperature of both animals showed a distinct rise and both developed an eruption. Blood drawn from the hearts of both of these monkeys was used for the inoculation of other monkeys, some of which developed febrile reactions and eruptions. This strain of measles virus was successfully propagated by blood inoculations through six monkey generations, when it was discontinued. The results obtained from the inoculations made from our fourth patient plainly showed us that the monkey was susceptible to measles by blood inoculations, but that this susceptibility was not acute and apparently was subject to quite considerable individual variation.

Since our work was reported three papers by different workers have appeared corroborating our results as to the susceptibility of the monkey to measles. Hektoen and Eggers,[5] while mainly occupied in their work on experimental measles in the monkey with a study of the leukocytes, state that the general results of their experiments agree very well with those reported by Anderson and Goldberger.

Nicolle and Conseil[6] reported the infection of the bonnet monkey with measles by the inoculation of blood drawn twenty-four hours before the first appearance of the eruption, and in a recent paper Lucas and Prizer also confirm our work as to the susceptibility of the monkey to measles by blood inoculation, and are the first to report the observation of Koplik spots in the monkey.

LIMITATION OF INFECTIVITY

The results obtained by these workers emphasize the statement made by us in our second paper[4] as to the variation in susceptibility of monkeys to measles, but the variability in susceptibility of the animals to the disease while apparent early in our work, did not, in our opinion, fully explain the uncertain results obtained from the inoculations made from our first three patients. For that reason, after we had obtained such striking results from the inoculations made from the fourth patient, we carefully went over the notes of our first three cases to determine if possible whether this question of the susceptibility of the monkey was the only factor, or if there was not some other factor having something to do with the inconclusive results from our first three cases.

---

5. Hektoen, Ludvig, and Eggers, H. E.: Experimental Measles in the Monkey with Special Reference to the Leukocytes. Jour. Am. Med. Assn., Dec. 2, 1911, p. 1833.

6. Nicolle, Ch., and Conseil, E.: Reproduction expérimentale de la rougeole chez le bonnet chinois. Virulence du sang des malades 24 heures avant le début de la éruption. C. R. de Acad. Sci., Dec. 26, 1911, cliii.

We found that the blood from our first three patients had been drawn after intervals of at least twenty-four to forty-two hours after the first appearance of the eruption, whereas the blood from our fourth patient had been drawn at the end of not less than fourteen hours after the eruption first appeared. The thought at once occurred to us that in measles we might have a disease in which the infectivity of the blood was more or less limited to the early period of the disease, as is the case, for example, in yellow fever, and that this limitation of infectivity of the blood was one of the factors concerned in the inconclusive outcome of our first three experiments.

In order to test this hypothesis, we drew blood from a patient with measles about six hours before the eruption first appeared. The patient was again bled exactly twenty-four hours later, or about eighteen hours after the first appearance of the eruption; the third bleeding was forty-five hours after the second, and the fourth forty-eight hours after the third. At least two monkeys were inoculated from each bleeding as soon thereafter as possible. Both of the animals inoculated with blood drawn before the eruption appeared, gave a definite febrile reaction but no eruption; all three of the animals inoculated with the blood (one with serum, one with washed corpuscles, and one with defibrinated blood plus normal monkey blood) drawn eighteen hours after the first appearance of the eruption, gave well-marked reactions, including the presence of an eruption and involvement of the respiratory apparatus; no evidence of a reaction in one and at most a very doubtful reaction in the second of the two animals inoculated with blood drawn sixty-five hours after the eruption first began to appear; no indication of a reaction in either of the two monkeys inoculated with blood drawn 113 hours after the first appearance of the eruption.

These results taken with those reported by other observers since our work first appeared, point strongly to a period of infectivity of the blood beginning at least twenty-four hours before and continuing for about twenty-four hours after the first appearance of the exanthem. At the end of twenty-four hours from the first appearance of the eruption the infectivity of the blood for the rhesus monkey appears to be greatly lessened and becomes progressively less thereafter.

The existence of a more or less definitely limited period of infectivity of the blood in measles and the variation in susceptibility of the monkey to the disease would explain both the negative and positive results of Josias, would throw light on the inconclusive results of some of our earlier experiments and the reported failure of other workers, and make understandable the opinion almost universally held heretofore that the monkey is not susceptible to measles.

NATURE OF THE REACTION IN MONKEYS

Before entering on a discussion of other results of our work with measles it would seem to be advisable to discuss briefly the nature of the reaction of the monkey to the disease. It has been shown that at least three species of the lower monkeys, *Macacus rhesus, Macacus cynomolgus,* and *Macacus sinicus,* are susceptible to measles, and it is quite probable that all monkeys are capable of taking the disease. Monkeys may be infected by introducing the virus into the body by any one of the usual methods of inoculation; but we have used, by preference, the intravenous method in most of our work. They also may contract the disease when placed in a cage with a sick monkey during, at least, the early period of the disease. When infection is produced by inoculation, the incubation period may be as short as five days or exceptionally as long as twenty-two days; but the average may be said to be approximately six to eight days.

The first indication of a reaction is usually a rise in temperature, especially a rise in the morning, with a sustained or higher reading in the afternoon; the temperature may remain at about the same level for four or five days and then drop rather sharply and remain down, unless there is a complicating bronchitis or pneumonia. The eruption usually begins to appear on about the third day after the rise in temperature begins, though it sometimes is coincident, or may be as late as the fourth or fifth day. In most cases it is first noticed on the chest or abdomen and may spread to the thighs, arms, and face. On account of the thick fur and dark skin the eruption is not so plainly discernible on the other portions of the body. It sometimes happens that the eruption begins to fade where it first appeared, while still appearing on other parts. As the eruption fades it may be followed by a fine branny desquamation. There is more or less variation, just as there is with human measles, in the appearance of the eruption; but the typical rash first appears as minute red spots which not infrequently coalesce, forming large patches; they soon begin to fade, but a stained spot may persist for several days. The eruption is not invariably present, but blood from monkeys with a distinct febrile reaction and with or without the respiratory symptoms has been found infectious. There is frequently coryza and coughing. In some instances the animal has been killed and found to have had a bronchopneumonia or even a lobar pneumonia. Some of our animals have presented as typical a picture of measles as will be seen in a well-marked case of the disease in human beings. Koplik spots have been observed by Lucas and Prizer.[7]

A monkey that has had a definite reaction is found to be immune to a subsequent inoculation with virulent blood.

---

7. Lucas, W. P., and Prizer, E. L.: An Experimental Study of Measles in Monkeys. Jour. Med. Research, April, 1912.

NATURE OF THE VIRUS

As before stated, the susceptibility of the rhesus monkey to measles appears to be subject to considerable variation, for it has not infrequently been found that when two animals were inoculated with the same material only one developed the disease, or that one gave a more marked reaction than the other.

As soon as we had definitely determined that the monkey was susceptible to measles by blood inoculation, we gave our attention to a study of the nature of the virus as it exists in the blood, and for this purpose made experiments to determine its filterability, resistance to drying, to heat, to freezing, and to age.

We found that the virus in measles blood is capable of passing through a Berkefeld filter, that it may resist desiccation for twenty-five and one-half hours, that its infectivity was destroyed by heating at 55 C., for fifteen minutes, that it resisted freezing for twenty-five hours, and possibly retained some infectivity after keeping twenty-four hours at 15 C.

NASAL AND BUCCAL SECRETIONS

In order to test the infectivity of the nasal and buccal secretions from human cases of measles, we obtained such secretions from a patient with the disease and made serial inoculations with these secretions at different stages of the illness. We found that the secretions from this case were infective for the monkey at the time of the first appearance of the eruption, and again forty-eight hours later; that is, early in the fourth day and early in the sixth day of the disease, and that this infectivity was due to a living virus susceptible of transfer from monkey to monkey by blood inoculation.

In order to determine the duration of the infectivity of the secretions, we made inoculations in monkeys with the secretions collected at various periods of the disease from five additional patients.

Without going into the details of these experiments it is sufficient for the purposes of this paper to say that, viewing our experiments with secretions as a whole, it would appear that the nasal and buccal secretions in uncomplicated cases of measles may be at times, but are not always, infective for the monkey, and that the positive results were obtained only with secretions collected in the twenty-four- and forty-eight-hour period of the eruption.

Our experiments so far do not permit us to draw definite conclusions as to the duration of the infectivity of the secretions, but we believe that our results tend strongly to suggest a reduction, if not a total loss, of infectivity of the nasal and buccal secretions with the approach of convalescence.

INFECTIVITY OF THE "SCALE"

We made three experiments[4] to determine the infectivity of the measles "scales" collected from human cases of the disease from four to seven days after the first appearance of the eruption. In not a single one of the monkeys — two in each experiment, or six altogether — was there the least evidence of a reaction. Alone, the negative results of these experiments, while highly suggestive, would hardly justify the conclusion that the desquamating epidermis is not infective for man; but, when viewed in the light of Mayr's negative results in children, we believe that the desquamating epithelium in measles does not, in itself, carry the virus of the disease.

CULTURES

We have made many cultures with measles blood known to be infective by monkey inoculations, but in no instance have we been able to obtain a growth that appears to have any etiologic relation to the disease.

THE DIAGNOSTIC VALUE OF THE CUTANEOUS TUBERCULIN TEST OF v. PIRQUET*

F. L. WACHENHEIM, M.D.
Attending Pediatrician, Sydenham Hospital and Mount Sinai Hospital Dispensary,
NEW YORK

Since v. Pirquet's[1] first publication, a vast amount of material has been collected concerning the anaphylactic (allergic) cutaneous test for tuberculosis. Most authors are inclined to the opinion that its value is limited; great during infancy, but during later childhood, as well as adult life, important only from a negative standpoint. It is admitted that a negative reaction is useful; it proves, except under certain special conditions that I need not refer to here, that the patient under examination has not been at any time infected with the tubercle bacillus. On the other hand, a positive reaction, after the earlier years of childhood, is believed to prove nothing save that such an infection has, at some time, occurred. It is generally accepted that the v. Pirquet reaction is as likely to be positive in a case of healed tuberculosis, as when the disease is still active. The chief argument to this effect is its occurrence in a very large proportion of apparently healthy older children and adults.

I shall here attempt to show that this adverse opinion is far too sweeping, that the value of the test has been underrated, and that the significance of a positive reaction is nearly as great in later childhood as in infancy, at least under conditions such as I have encountered.

I shall begin by reviewing some of the adverse evidence. Heim and John[2] found that 60 to 80 per cent. of all children above the age of infancy gave a positive reaction. Hamburger[3] found a percentage of 51 at 6 years, running up to 94 toward puberty. American statistics are somewhat more favorable; nevertheless, Shaw and Laird[4] report 45 per cent. positive after the age of 6. These data, which I could easily amplify, are certainly far from encouraging. Looking at the matter from a clinical aspect, however, the situation appears more promising; thus, Gewin,[5] while admitting that 50 per cent. of suspected cases give a positive reaction, shows that the ratio falls to 3 per cent. among patients not suspected

*Read before the Metropolitan Medical Society, New York, April 23, 1912.

1. Wien. med. Wchnschr., 1907, No. 38.
2. Wien. klin. Wchnschr., 1908, No. 8.
3. Wien. med. Wchnschr., 1909, No. 25.
4. Arch. Pediat., July, 1909.
5. Nederl. Tijdschr. v. Geneesk., 1908, v. 2.

of tuberculosis. The data of Wolff-Eisner and Brandenstein[6] are not quite so striking; they give a postive reaction in about one-tenth of clinically non-tuberculous infants and children. More recently, Feer[7] under similar conditions, obtained a still higher ratio, namely, 16 per cent.

The above discrepancies are sufficient to suggest that the personal equation has played a great part in these investigations, for even the last three sets of data cannot be reconciled. Assuming that the testing of apparently healthy children is purely academic, and that its results are clinically unimportant, the investigations on suspicious cases are in great need of revision, though their results have been in somewhat better accord with the clinical findings.

The following investigations, made at the Mount Sinai Hospital dispensary, include fifty cases, that fall into two groups. The first group includes children who were evidently or probably infected with tuberculosis, judging from the physical examination or symptomatology alone. The second group includes such as were below normal, but did not warrant a clinical diagnosis of tuberculosis; these children suffered from chronic cough, malnutrition, glandular enlargement, and other affections known to be frequently associated with tuberculosis, thus forming a series of cases in which some bacteriologic test should be of especial value.

My first group includes only thirteen cases, six manifestly and seven probably tuberculous. Only three were under 7 years of age. In all these cases the reaction was positive, and in four of the first six it was very intense, whereas all the other cases save one reacted only moderately. Without endeavoring to form a final conclusion from so small a material, it seems as if the intensity of the reaction stands in close relation to the severity of the infection. The chief point, of course, is the observation that not one case of certain or probable tuberculosis failed to react, and all authors are agreed that in chronic tuberculosis the proportion of negative reactions is exceedingly small.

Far more interesting is the second group, embracing thirty-seven cases, in which the children were subjected to the test because of persistent bronchitis, poor nutrition, slight adenopathy, doubtfully rheumatic joint affections, a tuberculous family history, and various other conditions that might possibly be referred to an infection with tuberculosis. The age classification of these children was as follows: Nine were under 5 years of age, the other twenty-eight ranging from 5 up to 13. Among these thirty-seven cases only two reacted positively, 5.4 per cent., a ratio almost as low as any hitherto reported, and far below the figures of Wolff-Eisner and Feer. It might be conceived that some of the discrepancy to the disadvantage of the latter is due to the inclusion of doubtful reactions,

6. Mitt. a. d. Grenzgeb. d. Chir. u. Med., 1908, v. 19.
7. Beitr. z. Klin. d. Tuberkulose, 1910, v. 18.

but Feer happens to be particularly exacting on this point. I always follow the method that I learned from v. Pirquet himself, scarifying in three places, with the control test in the middle, and using undiluted old-tuberculin. I do not see the patient again for forty-eight hours, and then record even a moderate reaction as positive. Thirty-five of my patients, therefore, did not react in the faintest degree, a most important point. I pay no attention to a reported slight reddening on the first day, which has disappeared on the second, and am sustained in this view by Feer and others, though as yet no universal agreement exists on this point. The typical reaction is quite unmistakable, persisting for a week at least. In what I call an intense reaction the erythema surrounding the papule is a half-inch in diameter, and persists for a very considerable time, even up to a month.

Confusion may arise from the recent modification of making only two scarifications. As the discomfort of the operation is trivial, I regard its curtailment in any respect as a technical error, since uncertainty should be avoided at any cost. Thus, for example, Bernheim-Karrer[8] says that children suffering from eczema give a positive reaction. This supposed reaction may very well be due to an infection from scratching, as occurred in a nephritic patient in my service at Sydenham Hospital. In this case one of the lateral scarifications remained free, whereas a papule formed on the control test; I may add that the lesion was not exactly like the tuberculin papule, but the resemblance was nevertheless close enough to have led to confusion if the location of the papules had been different. In a day or two these lesions became excoriated; this is not a regular feature of the tuberculin papule, but nevertheless does not exclude it. I repeat that it is safest to scarify according to the methods proposed by the originator of the test.

Of the two positive cases in my second group, one patient was an Italian, a point to which I shall recur presently; the other was the sister of one of my positive patients, who herself presented no suspicious symptoms. In another family, the brother of a positive patient gave an absolutely negative reaction. It is therefore difficult to exaggerate the value of the test. Its positive as well as negative aspects are of almost equal and very great utility in diagnosis; nevertheless, the test is often regarded as beset with certain limitations, and I must discuss this side of the subject more fully.

While I have taken care to select especially unfavorable cases, excluding all children who were apparently healthy, so as to raise the positive ratio as high as possible, still my figures remain uncommonly low. I account for this by recognizing that my statistics refer chiefly to a population that is notoriously refractory to tuberculosis, and is particularly

8. Cor.-Bl. f. schweiz. Aerzte, 1910, No. 31.

free from bovine tuberculosis because of its dietary laws and the prevailing practice of boiling milk. Klose[9] has shown that the cutaneous test furnishes no means of differentiating between the two types of tuberculosis; evidently, therefore, the relative rarity of the bovine type among my clientele would materially reduce my figures. The racial feature also is of some importance. In the children's ward of Sydenham Hospital, where many of the patients are of south Italian origin, my results have been somewhat different. While my observations there do not as yet embrace a large material, I have nevertheless noted a relatively high frequency of positive, and even intense, reactions in Italian children who were not even suspected of tuberculosis.

It would be rash to draw any conclusions from so fragmentary a study, but it seems fair to recognize that tuberculosis, latent or semiquiescent, is far more frequent among Italian than Jewish children, and that the considerable difference in the results obtained from these two sources forms no argument against the diagnostic value of this test, but, on the contrary, emphasizes it. A positive reaction should be regarded as an important danger signal, as evidence that a tuberculous process is either still active, or likely to become active at any time, and never to be carelessly dismissed as a mere relic of a past and gone infection. Still more erroneous is the assumption that most persons, well or ill, give a positive reaction. My statistics show clearly that this is not at all the case, but that at least 90 per cent. of presumably healthy though not robust children above the age of 5 are free from any sign of present or past tuberculosis, save possibly among certain badly infected races or badly housed and fed communities.

In passing, I would make a brief reference to the Moro test, which consists in the inunction of tuberculin ointment. My experience, chiefly in private practice, shows it to be less reliable than the v. Pirquet test, as it is often negative when the latter is positive. I cannot agree with those observers who consider the v. Pirquet test too delicate for practical diagnosis; my own statistics convince me that this is not the case; of all tests it is the least equivocal and the easiest and most accurate in its application.

CONCLUSIONS

The conclusions to be derived from the above data are obvious.

If 95 per cent. of all more or less run-down children give a negative reaction, a positive reaction in the small remainder becomes highly significant. Even an older child that responds to the cutaneous test should be regarded seriously, and by no means as one of the common run; an energetic antituberculous prophylaxis should be promptly initiated, and the patient should be guarded, so far as possible, with our

9. Deutsch. med. Wchnschr., 1910, No. 48.

TABLE SHOWING ANALYSIS OF THE CASES INVESTIGATED

Age	0	1	2	3	4	5	6	7	8	9	10	11	12	13	Total
Clinical Diagnosis															
Pulmonary tuberculosis*	..	..	..	..	..	..	..	1	1	..	..	2	..	..	4
Coxitis*	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1
Tuberculosis of wrist*	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Pulmonary tuberculosis(?)*	..	..	..	..	..	..	..	..	2	1	1	..	..	..	4
Tuberculous adenitis (?)*	..	..	..	..	1	..	..	..	..	..	1	..	..	..	2
Tuberculous peritonitis (?)*	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1
No tuberculosis !!!	..	..	..	..	..	..	..	..	..	..	1	..	1	..	2
Persistent bronchitis†	..	..	3	1	..	2	2	3	2	..	3	1	..	1	18
Malnutrition†	1	..	..	..	..	2	..	2	2	1	..	..	..	..	8
Chronic colitis†	1	..	..	..	..	..	..	..	..	..	1	..	..	..	2
Exudative diathesis†	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Chronic adenitis†	..	..	..	..	..	1	..	1	..	..	..	..	..	..	2
Pleurisy†	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Rheumatism†	..	..	..	1	..	..	..	..	..	..	..	1	..	..	2
Unresolved pneumonia†	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1
Total Male	2	1	2	1	2	2	2	4	3	0	4	2	0	1	26
Total Female	1	1	1	1	0	3	0	3	5	2	4	2	1	0	24
Grand total	3	2	3	2	2	5	2	7	8	2	8	4	1	1	50

*Reaction positive. †Reaction negative.

somewhat limited resources, against the recrudescence of a disease which it is safest to regard at the best as latent, and by no means extinct.

Our acquaintance with the v. Pirquet test is too brief to inform us as to the relative frequency of active tuberculosis after adolescence, in individuals who seemed fairly healthy, but reacted positively to the cutaneous test in childhood. The positive determination of the tuberculo-anaphylactic state is therefore of the greatest diagnostic value, and should make our remote prognosis extremely guarded.

I append an analytical table of the material investigated.

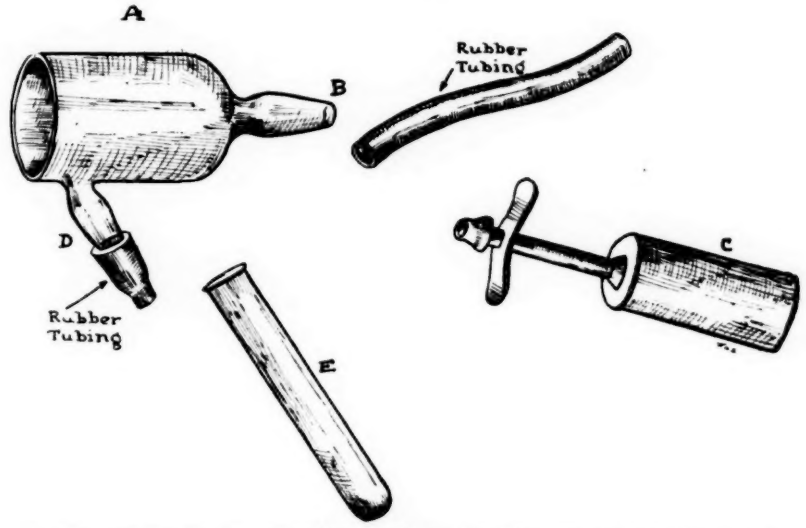
111 West Eighty-Fifth Street.

APPARATUS FOR COLLECTING INFANTS' BLOOD FOR THE WASSERMANN REACTION *

KENNETH D. BLACKFAN, M.D.
ST. LOUIS

The methods usually employed for obtaining blood from infants are in the majority of instances extremely unsatisfactory. The veins are too small to enter and it is a tedious, difficult and painful procedure to collect the necessary amount of blood by puncturing the fingers or toes.

In European clinics use is made of a suction apparatus. This, in our hands, has proved unsatisfactory in its application to very poorly nourished infants and it is unnecessarily elaborate. The modification herein described is a simple, inexpensive device by which the necessary amount of blood can be obtained easily, with a minimum of discomfort



Suction apparatus for collecting blood for the Wassermann reaction. A, suction glass connecting at B with suction pump, C. The blood flows through D, connected by rubber stopper with an ordinary test-tube E.

to the patient and in a comparatively short space of time. The apparatus has been in daily use at the St. Louis Children's Hospital and the time spent in collecting the blood averages about two minutes.

The apparatus, as shown in the accompanying illustration, consists of a glass cylinder (A) 1⅝ inches in diameter; the large end is ground

* From the Department of Pediatrics, Washington University, St. Louis, Mo.

smooth and the other end (B) is drawn out for the attachment of a small hand suction pump (C); an outlet (D) which is fused at an angle to the under surface of the cylinder, one-half inch from the large end, and a collecting tube (E). The collecting tube (E) fits over the outlet (D), the connection being made air-tight by means of a small piece of rubber tubing. The tube in which the blood is collected may be the size which is used in the laboratory test, obviating the necessity of transferring the blood to another tube.

TECHNIC

The technic is as follows: Connect the apparatus for use. The patient may be placed in either the upright or recumbent posture. The most convenient site is on the back just below the angle of the scapula, though any other surface of the body may be used. Having cleansed the area with alcohol and ether, one or two small punctures are made through the skin with a sharp-pointed scalpel or Hagedorn needle and the apparatus is quickly applied. With but little suction force the blood will flow from the wound through the outlet and into the collecting tube. After a sufficient amount has been collected, the tube is taken off, a cotton plug is inserted and the tube containing the blood is set aside until the examination is made. The wound is covered with a collodion dressing and the resulting swelling rapidly subsides with no discomfort to the patient.

Blood may be collected by this method from older children and adults, as well as infants, in a much shorter time and with less difficulty than by entering a vein.

The glass apparatus can be made in the laboratory or by a glass blower and the suction pump can be obtained through any surgical supply house.

1806 Locust Street.

TETANUS AS A COMPLICATION OF BURNS*

CHARLES NEWBERGER, B.S., M.D.
CHICAGO

The coincidence of two cases of burns, each followed by tetanus, occurring within two days of one another, coupled with the prevalent belief as to the supposed infrequency of such a complication, formed the incentive for this report and review of the literature.

CASE 1 (Reported by courtesy of Dr. J. A. Graham).—E. F., female, 6 years old, admitted to the Children's Memorial Hospital August 23, 1911, with history that about three hours before while playing about a bonfire, she jumped too near the flames, caught her clothes on fire, and sustained a burn of the body.

Examination.—Burn of first degree, and, to a slight extent, of second degree, covering all of back from neck to buttocks, and extending around the right side to the middle line in front. Burn also of both hands, and of right upper arm. Child very nervous and irritable; cried and talked almost constantly, and at times was delirious. Pulse 112, temperature 98.8 F., respiration 24. Urine: slightly cloudy, pale color, sp. gr. 1030, acid, with trace of albumin, several hyaline casts, and a few red and white blood cells, and some epithelial cells. White blood count showed 9,500 per c.mm. Vaginal and throat smears and throat culture were negative.

Treatment.—Local dressings with picric acid, later changed to sterile vaselin with ¼ per cent. phenol (carbolic acid); strychnin sulphate, 1/100 gr. hypodermatically every eight hours; brandy m. xxx, every eight hours; soft diet, later changed to rectal feedings every six hours of 8 ounces of peptonized milk. Morphin suppository, gr. 1/12, when necessary for pain.

Course.—The child got along fairly well for about a week; the temperature ranged from 99 to 103 F.; pulse varied from 90 to 130, and respiration 25 to 40. Delirium became less pronounced, child took her nourishment, and the local condition began to improve. August 30, the eighth day after the accident, the child complained of rigidity of the jaw; later, at night, she had difficulty in swallowing. About 10 the next morning, on attempting to swallow, the jaw became very rigid, teeth set, back curved upwards, limbs extended, lips very cyanotic, and respiration stertorous and difficult. The convulsion lasted about two minutes, and recurred about every twenty minutes. At 10:50 a. m. 3,000 units of tetanus antitoxin was injected, but the convulsions were frequently repeated, the child became more cyanosed, foamed at the mouth almost constantly, and at 2:10 p. m. died in convulsions, the temperature shortly before death being 103.2 F., pulse 160, and respiration 50.

CASE 2 (Reported by courtesy of Dr. C. A. Parker).—E. H., female, 5 years old, admitted to the Children's Memorial Hospital Aug. 25, 1911, with a burn sustained a half hour before, while playing about a fire. Passers-by, in trying to extinguish the flames, dragged the child several feet on the ground.

Examination—Burn over all of right side, front and back, extending from face and neck down to middle of thigh, the greater part being of superficial extent, and in places, of second degree. Temperature 100 F., pulse 124, respiration 28. The child was not disturbed for further examination. Urine: acid,

*Read before the Chicago Pediatric Society, May 21, 1912.

slightly cloudy, amber in color, sp. gr. 1026, with a trace of albumin, a few hyaline casts, some epithelial cells, and a few red and white blood-cells. Blood count showed a leukocytosis of 29,300. Vaginal and throat smears, and throat culture were negative.

Subsequent History.—The child was placed in a ward away from Case 1, and in charge of other nurses. Dressings of sterile vaselin were applied, morphin suppository, gr. 1/12 given for pain, aromatic cascara ordered, and liquids by mouth. The child did not sleep well, and for 24 hours passed no urine, and then only 3 ounces. The following day, August 26, the parents took the child home against advice. On September 1, the child was brought back to the hospital. The burns had been dressed every day by the family physician, fever had been present every day, child had had some constipation, later there was diarrhea, some swelling of right leg and foot, and at noon on the eighth day after the accident she complained of pain on opening the mouth. Examination showed the jaws set; the teeth could be separated only about ⅛ of an inch; the child swallowed with difficulty, and some of the milk came out through the nose. At 8:40 p. m. 3,000 units antitetanic toxin was given. Child had a restless night. The next day, at 9:15 a. m., convulsion occurred, and 3,000 more units of antitoxin was injected. Her general condition became worse, attacks coming on frequently, with rigidity, difficulty of respiration, and marked cyanosis. At 2:20 p. m. a convulsion set in with twitching and jerking movements of arms and legs, rigidity of back, firmness of jaws, prominence of muscles of neck, hissing and foaming of mouth, and cyanosis; consciousness was present, although convulsion was continuous, respiration becoming more difficult, more shallow, and less frequent until death occurred at 3:28 p. m.

These two cases are noteworthy because of their coincidence, similarity in manner of occurrence, course, and result. In each instance, the symptoms of tetanus set in on the eighth day after the burn, and the complication proved fatal about twenty-six hours after its appearance.

A review of the literature available shows that tetanus is not an infrequent occurrence after burns, for forty-seven cases are reported with more or less detail, and mention is made of about twenty others; these, with the two reported above, make a total of sixty-nine cases. Those which are mentioned only, include two cases by Rose,[1] four by Taylor,[2] two by Lockwood,[3] eleven by Norris,[4] and one by Billroth.[5]

A tabulated summary of the reported cases is given herewith.

From a study of the data furnished by these forty-nine cases, the following facts are noted:

With Reference to Season.—Tetanus may arise as a complication of burns during any month of the year, but it is more common during the colder months. The mortality rate, however, is somewhat higher in the warmer months. From November to April, inclusive, occurred 74 per cent. of the cases, with a mortality of 73 per cent.; 26 per cent. of the series came in May to October, inclusive, and of these 83 per cent. died.

Sex.—The condition is met with equal frequency in the two sexes. The chances for recovery are one in five for females, and one in four for males. Of the forty-nine cases, twenty-five were in females, with five recoveries, and twenty-four in males, with six recoveries.

Age.—The complication may present itself in patients of all ages; the youngest case recorded is that of a boy of 4 years, the oldest is that of a woman 64 years of age. It is most common between the ages of 11 and 20. Eighteen per cent. of the cases occurred during the first decade, about 33 per cent. during the second decade, 26 per cent. in the third, 18 per cent. in the fourth, and 4 per cent. in the sixth. It proved fatal in all those above 35 years of age, in 87 per cent. of those between 11 and 20, and in about 75 per cent. of the remaining ages.

Location of Burn.—In 40 per cent. the trunk was involved together with the upper, or lower, or all of the extremities; in 25 per cent. the lower extremities only; in 17 per cent. the upper only; in 10 per cent. the trunk alone, and in 6 per cent. the head and face. The mortality is greatest when tetanus complicates burns where the trunk is involved, either alone, or with burns of other parts of the body, for 87.5 per cent. of the patients so burned died. When either the upper or the lower extremities are injured, or the head and face alone, the chances of recovery are about one in three.

Period of Incubation.—The interval between the date of burn and the onset of tetanic symptoms varied from twelve hours in one instance to four weeks in two other cases. In thirty-three cases (67 per cent.) the complication appeared within the first twelve days. Of these, more than 90 per cent. were fatal. In the remaining sixteen cases, tetanus set in after the twelfth day, and eight (50 per cent.) recovered. Of four cases coming on after the third week all got well.

Duration.—The complication may run either an acute or chronic course; the extremes noted in this series are five hours in one case and six weeks in another, but the majority terminate within ten days. Of the forty-nine cases, thirty-seven (75 per cent.) ended within the first ten days after the onset of the tetanic infection, and of these, sixteen cases (32 per cent.) terminated within forty-eight hours. Of the thirty-seven cases, all but one died; in the remaining twelve cases, the course ranged from two to six weeks, and ten patients recovered.

PROGNOSIS

Of the forty-nine patients, thirty-eight died and eleven recovered, making a mortality rate of 77 per cent. Death usually occurs within the first week after tetanus makes its appearance. The prognosis is less favorable in cases occurring in the warmer months of the year, in adult life, where the trunk is involved, and when the incubation period is short (less than twelve days).

SYMPTOMATOLOGY

Tetanus may set in after a burn, regardless of its location, extent, or degree. The earliest and most frequent symptom is a stiffness of the jaws with a consequent difficulty in opening the mouth and in swallowing;

SUMMARY OF REPORTED CASES OF TETANUS FOLLOWING BURNS

Case	Date	Sex	Age	Part of Body Involved	Incubation*	Duration*	Result	Remarks
1	Oct. 10, 1836[6]	F	13		13	6	D	
2	April, 1841[7]	M	32	Feet	30?	21	R	Assafetida every 2 hrs. gr. v, by mouth, or ½ oz. of tr. per rectum
3	Nov. 6, 1846[8]	F	9?	Right lower extremity (deep)	10	36 hr.	D	
4	Nov. 10, 1846[8]	F	25	Right arm (superficial)	18	8	D	
5	Dec. 28, 1846[9]	F	19	Upper extremity and back (superficial)	9	5	D	
6	Apr. 10, 1848[10]	F	30	Back (superficial)	16	4	D	
7	Jan. 30, 1849[11]	F	12	Trunk, face and arms (superficial)	9	5	D	
8	Feb. 1, 1849[12]	F	17	Waist and left lower extremity (severe)	8	36 hr.	D	
9	Jan. 25, 1852[13]	F	8	Chest and arms (severe)	15	3	D	During a paroxysm, glottis remained so long closed that laryngotomy was performed
10	Nov. 15, 1852[6]	F	11	.....	9	4	D	
11	Feb. 15, 1853[13]	F	10	Trunk and right arm (severe)	11	10 hr.	D	
12	Feb., 1853[14]	M	4	Head, chest, left upper extremity (severe)	9	14	R	Dover's powd. gr. 2 Sesquiox. iron gr. 4 every 4 hrs.
13	Oct. 7, 1853[15]	F	36	Right buttock (superficial)	12	3	D	
14	Dec. 4, 1853[16]	F	64	Trunk and lower extremities (severe)	8	14 hr.	D	Epileptic
15	Jan. 22, 1854[15]	M	12	Trunk and arm (severe)	12	64 hr.	D	
16	Feb. 9, 1854[17]	F	9	Face and arms	7	24 hr.	D	
17	Feb. 19, 1855[13]	F	38	Right lower extremity (severe)	12	6	D	
18	Apr. 5, 1855[18]	F	9	Upper trunk and arms	9	5 hr.	D	
19	—, 1858[1]	M	25?	Lower trunk (severe)	16h	6 hr.	D	
20	Oct. 16, 1859[19]	M	24	Left arm, chest, and hip	11	3	D	Epileptic
21	Mar. 19, 1861[1]	M	34	Thighs (severe)	4?†	14	R	
22	Apr. 9, 1861[20]	M	18	Arms	10	3	D	
23	Apr. 9, 1861[20]	M	16?	Arms	20	?	D	
24	Jan. 14, 1862[1]	M	32	Lower extremities (severe)	11	2	D	
25	Feb. 19, 1866[21]	M	40	Chest and lower extremities (severe)	9	20 hr.	D	Alcoholic
26	Apr. 2, 1869[22]	M	12	Lower extremities	12	7	D	
27	Jan. 1, 1874[2]	M	31	Head	10	6	D	Epileptic

*In days, unless otherwise specified. †Weeks.

SUMMARY OF REPORTED CASES OF TETANUS FOLLOWING BURNS—(*Continued*)

Case	Date	Sex	Age	Part of Body Involved	Incubation*	Duration*	Result	Remarks
28	—, 1875[2]	F	14	Back and thighs (severe)	9	24 hr.	D	
29	July 1, 1875[2]	M	18	Left elbow (superficial)	4	16	D	
30	June 9, 1877[23]	M	22	Head, face, arms chest (severe)	9	2	D	
31	Nov. 28, 1878[24]	F	12	Trunk and extremities (severe)	7	8 hr.	D	T. 104.6 F. at time of death
32	Dec. 22, 1878[25]	F	26	Left hand and chest (severe)	8	20	R	Epileptic. Amputation of hand
33	Dec. 5, 1882[26]	M	22	Extremities (severe)	18	36	R	Large doses of KBr, chloral hydr. and morphin.
34	Dec. 9, 1886[27]	M	12	Extremities (superficial)	4	10	D	
35	Jan. 1, 1887[28]	F	24?	Left leg, arm, and thorax (severe)	21	42	R	Frequent doses of morph. acet. 87 gr. in 35 days
36	—, 1889[29]	M	60	Knees (severe)	14	60 hr.	D	
37	Feb. 20, 1893[30]	M	30	Right leg and foot (severe)	8	7	D	Venesection. Behring's serum, 4 injections, 20 gr. each
38	Nov. 2, 1894[31]	M	9?	Leg and forehead (slight)	14	21	R	Tizzoni antitoxin, 12 injections, 5 and $\frac{3}{8}$ gm.
39	Nov. 12, 1894[32]	M	15	Lower extremity (deep)	11	10	D	Six injections of antitoxin, 111 gr.
40	June 26, 1895[33]	M	39	Chest and extremities (severe)	10	42 hr.	D	
41	Dec. 27, 1898[34]	F	12	Right arm and axilla	14	15	R	12 injections in 9 days of 110 c.c. antitoxin
42	May 7, 1900[35]	F	40	Trunk, face, arm (superficial)	19	36 hr.	D	
43	June 17, 1900[36]	M	30	Left lower extremity (severe)	21	8	R	7 m. phenol q. 3 hrs. subcutaneously
44	Feb. 23, 1902[37]	M	30	Chest	12	4	D	Intracerebral injection of 3 cm. serum
45	Dec., 1906[38]	F	25	Left arm (slight)	12	17	R	2 c.c. of 2 per cent. phenol sol. hypo. for 10 days, every 4 hrs.
46	June 6, 1909[39]	F	19	Face, chest, upper extremities (severe)	19	25	R	Antitetanic toxin; lumbar puncture
47	June 20, 1909[39]	M	24	Head, face, trunk, arms (severe)	17	4	D	
48	Aug. 23, 1911[40]	F	6	Trunk and upper extremities (superficial)	8	26 hr.	D	Antitetanic toxin
49	Aug. 25, 1911[40]	F	5	Right side (superficial)	8	26 hr.	D	Antitetanic toxin

*In days, unless otherwise specified. †Weeks.

this is soon followed by pains and rigidity or spasms of the muscles of the neck, back, abdomen, or limbs. Within twelve to twenty-four hours, there may occur opisthotonos, risus sardonicus, cyanosis, and profuse sweats. In children, delirium and restlessness may be present. Fever is always noted, and in some instances may be very high just before the fatal termination (107.1 F. in Case 44), and it may persist for two hours or more after death (104.7 F. two hours after death in Case 37). The respiration is rapid, labored, and shallow, and the pulse small and frequent, especially toward the end. Consciousness may remain undisturbed until death ensues. This was noted in five patients, including one of the author's cases. There may be constipation. The urine shows no marked changes; the blood may be normal or present a leukocytosis. Death occurs from exhaustion, or because of spasm or paralysis of the muscles of respiration.

Post-mortem examination of those cases which came to autopsy revealed no unusual pathologic findings.

TREATMENT

Aside from the local treatment of the burn, alleviation of pain, attention to the bowels, and the use of supportive measures, it would be well, in view of the possibility of tetanic infection, to give patients in all cases of burns a prophylactic injection of antitoxin. None of the cases reported were so protected against the complication. When tetanus has set in, the value of antitoxin may be questioned. In this review, of the eight cases (37, 38, 39, 41, 44, 46, 48, and 49) in which the patients were treated with antitetanic serum, only three recovered (Cases 39, 41, and 46). As the incubation period in two of these cases was fourteen days, and in the third, nineteen days, and as it has been shown that in cases developing after the twelfth day the patients have an even chance of getting well regardless of the treatment employed, the part played in these recoveries by the serum is rather uncertain.

Two patients were treated with phenol, and both recovered; one (Case 43, incubation of twenty-one days) received subcutaneous injections of 7 minims of phenol every three hours, for eight days, some relief being obtained after four doses; the other (Case 45, with tetanus twelve days after the burn) was given 2 c.c. of a 2 per cent. solution of phenol hypodermatically (Baccelli's method) every four hours for ten days.

Two cases recovered under large doses of morphin; one of these (Case 33, incubation eighteen days) got as much as 6 gr. of morphin sulphate a day, and the other (Case 35, incubation twenty-one days) received morphin acetate in one-fourth to 1 gr. doses, taking from 1¾ to 6½ gr. in twenty-four hours, and in thirty-five days, a total of

87 1/6 gr. Another patient (Case 32, incubation eight days) had an amputation of the infected hand performed six days after the onset of tetanus, and the woman recovered.

CONCLUSIONS

1. Tetanus is not an infrequent complication of burns.

2. The mortality is greater in cases occurring in the warmer months of the year.

3. Tetanus complicating burns involving the trunk is more serious than when the burn is on other parts of the body.

4. The longer the incubation period the less the mortality.

5. The greatest number of fatalities occurs early in the complication.

6. The prophylactic injection of tetanus antitoxin is advisable in all cases of serious burns.

3416 Douglas Boulevard.

REFERENCES.

1. Rose, E.: "Ueber den Starrkrampf."
2. Taylor, F.: Guy's Hospital Reports, 1878, xxiii.
3. Treves, F.: "System of Surgery," 1895, i.
4. Norris, G. W.: Phila. Med. Jour., May 16, 1903.
5. Billroth: "Allg. Chir. Path. u. Therap.," 1906.
6. Guy's Hospital Reports, Series 3, 1857, iii.
7. Am. Jour. Med. Sc., 1841, ii, 344.
8. Med. Times and Gaz., 1854, i, 435.
9. Ogle, J. W.: Brit. and For. Med.-Chir. Rev., 1868, xli, 491.
10. Watson, J.: Tr. N. Y. Acad. Med., 1857-1863, ii, 207.
11. Med. Times and Gaz., 1854, i, 489.
12. Med. Times and Gaz., 1854, i, 488.
13. Butcher, R. G. H.: Dublin Quart. Jour. Med. Sc., 1855, xix, 307.
14. Med. Times and Gaz., 1854, i, 436.
15. Med. Times and Gaz., 1854, i, 376.
16. Med. Times and Gaz., 1854, i, 490.
17. Med. Times and Gaz., 1854, i, 406.
18. Wilks, S.: Guy's Hosp. Rep., S. 3, 1856, ii.
19. Ogle, J. W.: Brit. and For. Med.-Chir. Rev., 1868, xli, 507.
20. Clark, F.: Med. Times and Gaz., 1861, i, 672.
21. Gadaud: Bull. de la Soc. anatom. de Paris, 1866, xi, 540.
22. Watson, E.: Practitioner, 1869, iii, 153.
23. Arch. f. klin. Chir., 1880, xxv, 381.
24. Guy's Hosp. Rep., S. 3, 1878, xxiv, 185.
25. Arch. f. Klin. Chir., 1880, xxv, 853.
26. Dougherty, A. C.: Med. News, Phila., 1884, xliv, 97.
27. Lewis, B.: St. Louis Med. and Surg. Jour., 1887, lii, 78.
28. Dukes, C.: Lancet, London, August, 1887, 405.
29. Koehler, A.: Charite-Annalen, 1889, xiv, 496.
30. Gessner, W.: "Ueber Tetanus." Inaugural Dissertation, Halle, 1899.
31. Tracey, H. E.: Lancet, London, February, 1896, 287.
32. Williamson, G. E.: Brit. Med. Jour., Feb. 23, 1895.

33. Godfrey, S. C., and Reed, J. L.: New Zealand Med. Jour., 1895, viii, 255.
34. Marshall, L.: Lancet, London, April, 1899.
35. Dubar: L'Echo med. du Nord, 1900, iv, 323.
36. Boehm, J. C.: St. Paul Med. Jour., 1903, v.
37. Suter, F. A.: Arch. f. klin. Chir., 1904-5, lxxv, 113.
38. Anderson, B. A.: Australasian Med. Gaz., 1907, xxvi, 123.
39. Michelson, F.: St. Petersb. med. Wchnschr., 1910, xxxv.
40. Author's case.

AN UNUSUAL LESION OF THE RIGHT CRUS IN AN INFANT OF FOUR MONTHS

FREDERIC J. FARNELL, M.D.
Neuro-serologist, Butler Hospital; Neurological Extern, Rhode Island Hospital, O. P. D.
AND
HENRY W. BURNETT, M.D.
Physician, Diseases of Children, Rhode Island Hospital, O. P. D.
PROVIDENCE

A case rather confusing to the practitioner, yet highly interesting because of its comparatively rare occurrence but far reaching effect, is that one of a young child brought to me for examination by Dr. Burnett.

History.—E. P., aged 4 months; Italian. The infant's mother and father were alive and well. One sister of the patient, 18 months old, was enjoying good health. The mother had had no miscarriages, the father denied specific infection and there was no history of tuberculosis in the family.

The child was born after a period of normal gestation and after an uneventful confinement. Labor was easy and of short duration, no instruments were used and there is no history of trauma. He had been fed on the breast and, with the exception of an occasional vomiting spell, which the mother attributed to "too much milk," the infant had had no grave physical disturbances.

The mother noticed that after the child was born the left eyelid drooped and that he did not move his left leg and arm as much as the right leg and arm. When three months passed without visible improvement the child was taken to Dr. Burnett, to whom I am greatly indebted for the privilege of examining this interesting condition.

Examination.—Physical examination showed a well-nourished child of four months with a ptosis of the left eye-lid and a left hemiparesis. He was able to move both his eye-balls in all directions. The pupils were unequal in size; the left being but a pin-hole which reacted to neither light nor accommodation but dilated quickly when one drop of 2 per cent. cocain was applied. The optic disks were normal. There was no involvement of other cranial nerves. Distinct weakness of the left arm, but more so of the left leg, was manifested. The left abdominal reflex was less active than the right and the left knee-jerk was stronger than the right. There was no ankle clonus. The Babinski sign was present on both sides (quite proper for a child of four months) and, so far as could be ascertained, sensation was normal.

During the two months previous to the preparation of this report the child had shown no material change. He was still in the same condition in which the mother saw him at birth and presented a left ptosis, a left immobile pupil and a left hemiparesis.

The difficulty in tracing these stigmata to the root lies in the fact that the exact anatomic relations of the individual nuclei of the oculomotoris are still a matter of dispute. That its complexity is not understood is not at all surprising when one recalls how large a number of muscles the third nerve innervates; how these muscles are anatomically and physiologically

dependent on each other and how close a relation they bear to other groups of nerves necessary to call forth proper co-ordination. Although studied by such noted investigators as Spitzka,[1] Starr,[2] Edinger,[3] Westphal,[4] Von Gudden,[5] Obersteiner,[6] Barker[7] and others, adequate information concerning these various parts has not been forthcoming.

The lower neurons of the oculomotoris possess cell bodies which are situated in the nucleus nervi oculomotoris located in the pons. On section it is seen to occupy a position in the tegmentum, ventral to the aqueductus cerebri, just dorsal to the longitudinal fasciculus and inclined at an angle to a line drawn parallel to the long axis of the fourth ventricle. The axons of each nerve are mainly from the nucleus of the same side, but also partly from the nucleus of the opposite side; that is, there is a partial decussation. This decussation contains the fibers mainly from the posterior or distal (spinal) third of the nucleus. The nuclei of the third nerve are very complex and consist of numerous groups of cells. Their appearance can be well shown by reconstruction such as that produced by Meyer or Sabin. By this reconstruction method the nuclei are seen to be in the shape of an arrow with its head ventralward and its shaft dorsalward.

In the case presented, interest lies mainly in the probability of a decussation of the third nerve by these decussating fibers. Perlia[8] and Von Koliker,[9] as well as Bernheimer,[10] have proven conclusively that there may be a decussation in man. Other investigators have shown that it exists in animals and birds. As a result many attempts have been made to localize the nucleus and root-fibers corresponding to individual eye muscles. The crossed fibers go through the fasciculus longitudinalis in its dorsal part and after a somewhat curved course pass near the lateral border of the red nucleus when, turning inward, they are traceable toward the medial and lateral portion. Starr found that the fibers to the ciliary muscles arose in the posterior portion of the medial group and that from

1. Spitzka, E. C.: The Oculomotor Centers and Their Coordinators. Jour. Nerv. and Ment. Dis., 1888, xv, 413.

2. Starr, M. A.: Ophthalmoplegia Externa Partialis; Jour. Nerv. and Ment. Dis., 1888, xv, 301.

3. Edinger: Verl. der central. Hörnervenbahnen, Arch. f. Psychiat. u. Nervenkr., 1885, xvi.

4. Westphal: Ueber die chronische progressive Lähmung der Augenmuskeln-Supply, Arch. f. Psychiat. u. Nervenkr., 1891, xxii.

5. Von Gudden: Gesammelte Abhandl., Wiesbaden, 1889.

6. Abersteiner: Anzeig. d. k. k. Gesellsch. der Aerzte in Wien., 1880.

7. Barker: The Nervous System, New York, 1901.

8. Perlia: Die Anatomie des Oculomotoriuscentrums beim Menschen. Arch. f. Ophth., 1889, xxxv; Sec. 287-308.

9. Von Kolliker: Sitzungsb. d. Phys-med. Gesellsch. zu Wurzb., 1892.

10. Bernheimer: Das Wurzelgebiet des Oculomotorius beim Menschen, viii, Wiesbaden, 1894.

the lateral portion arose the fibers to the levator palpebræ. On the other hand, Kahler and Pick stated that the pupillary fibers ran in the anterior bundles and that the fibers to the extrinsic muscles ran in a lateral and medial group. Schwabe,[11] who has attempted to trace out these fibers along lines suggested by Von Gudden, states that the fibers governing the levator palpebræ superioris and retrahens bulbi could not be connected with any cells. He is rather of the opinion that they are possibly innervated by cells in the most lateral dorso-distal group of the nuclei of the opposite side.

These notes, applied to the case above presented, permit argument. Since it presents a left ptosis, a left immobile pupil and a left hemiparesis the following inferences suggest themselves as possible explanations. First, we may here be dealing with an unusual case of an uncrossed pyramidal tract with a lesion in the crus in the region of the third nucleus. This deduction seems rather improbable when one recalls the exceedingly rare occurrence of such a condition. Second, there may be an agenesis, an aplasia or hypoplasia of the nuclei of the fibers that innervate the iris and Mueller's muscle. This agenesis would condition one lesion and the disturbance of the pyramidal tract would be another distinct condition. This seems far more probable because ageneses of the nuclei or portions of the nuclei are rather frequent occurrences, as is proven by pathologic examinations. Third, the lesion involves, and the one to which the writers feel more free to accept, the crossed fibers of the right third nerve in the posterior and distal portion, which is in close proximity to the pyramidal tract. Should this be so (and one of us is inclined to believe it is), the case would seem to justify Schwabe in his contention that it is possible that the fibers of the levator palpebræ come from the posterior and lateral portion of the nucleus of the opposite side and pass in close proximity to the pyramidal tract.

260 Benefit Street—274 Benefit Street.

11. Schwabe: Neuro. Centralbl., 1896, xv.

THE STANDARDIZATION OF BLOOD PRESSURE READINGS BY MEANS OF AN AUTOMATIC DEVICE FOR INDICATING SYSTOLIC AND DIASTOLIC PRESSURES IN CHILDREN*

B. RAYMOND HOOBLER, A.M., M.D.
NEW YORK

One cannot but be struck with the variation in systolic and diastolic blood-pressure readings on the same individual as recorded by different observers. In a recent test three separate observations were made on a series of ten patients, by three groups of observers working independently. The patients were in bed, did not change position between readings and the observations were all taken within half an hour. Each group of observers was supplied with a similar instrument. The first set used the older method of estimating systolic pressure by tactile pressure over the radial artery with the oscillation of mercury as the indicator for the diastolic pressure; the second group used the auscultatory method for both systolic and diastolic pressure, while the third employed the auscultatory method for systolic pressure and the Fedde pith-ball indicator for the diastolic. The observers were familiar with the apparatus, being accustomed to taking blood-pressures as a daily routine in the ward.

The results were astonishing. The widest variation in systolic pressure was 30 mm. mercury; that of diastolic, 40 mm. of mercury, while there was found a variation of 25 in the reading of mean pulse-pressure. In but few of the cases did two groups agree throughout and in none did all coincide exactly. The observers were equally experienced, none of them being experts in blood-pressure readings, but all had average ability.

It is evident, therefore, that with our present methods of blood-pressure reading at the bedside, there enters an element of variation which may be called the personal equation. In those observers who have served a long apprenticeship in the art of blood-pressure reading, this variation is small, while in those less expert it may be considerable.

In the every-day use of an instrument, this variation is approximately the same, and in comparative readings is of little consequence. But in absolute observations, especially those used for publication, this variation must be considered.

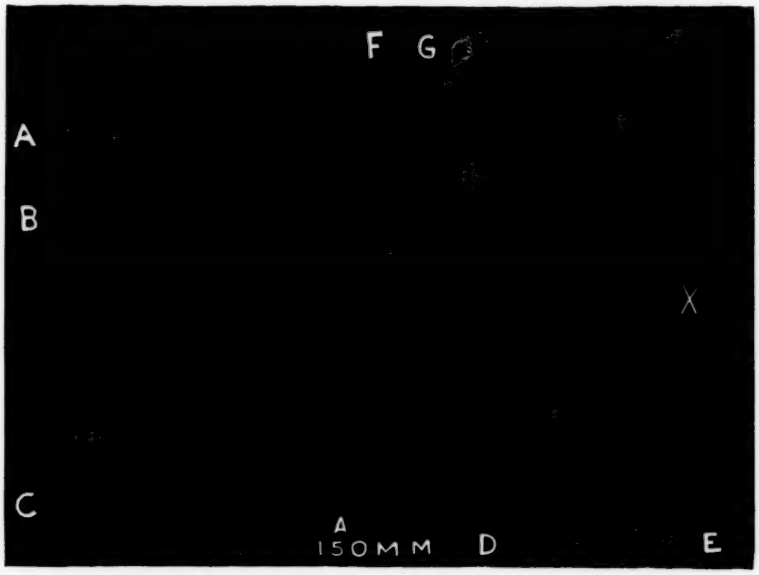
---

*Read in the Section on Diseases of Children of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

About a year ago I devised a method by which the systolic and diastolic pressures could be both visibly and automatically indicated, and published a description of it,[1] to which article those interested are referred for details.

During the past winter this instrument has been in constant use both in hospital and private work and has proved very reliable. In a long series of observations taken daily on many patients the variations have been extremely small, both in systolic and diastolic pressures, each day's observations being recorded without any knowledge of the previous day's readings.

The indicating device was tested against the readings of the Erlanger sphygmomanometer, the two instruments being connected in one circuit.



Blood-pressure tracing. A. Erlanger tracing. B. Plethysmograph tracing. C. Base line for indicating time of first movement of pith-ball. D, Point at which pith-ball first moved. E. End of experiment. F. Point of systolic pressure on Erlanger tracing. G, Point on Erlanger tracing at which pith-ball moved.

At the same time the arm was placed in a plethysmograph with recording needle leading to the tambour, on which was recorded the tracing of the Erlanger instrument. One of these tracings is herewith presented.

In the figure the uppermost tracing is from the needle of the Erlanger instrument, the middle one the plethysmograph, while the lowest line is that of the electric timer. On the last-named line is a break indicating the first movement of the pith-ball which occurred when the mercury

1. Hoobler, B. R.: Med. Rec., New York, December 30, 1911.

registered 150 mm. in the Erlanger instrument. Farther along on the base line is the point indicating the end of the experiment. If one measure back from this point on the base line to the break at 150 and likewise measure back the same distance on the tracings of both the Erlanger instrument and of the plethysmograph, one will note the coincident phenomena indicated by them. It will be seen that the amplitude of the tracing of the Erlanger instrument has widened considerably, indicating that the point of systolic pressure is found slightly earlier by the Erlanger tracing than by the pith-ball indicator. On the plethysmograph tracing it will be noted that there is a gradual raising of the lever as the arm fills with blood. In six consecutive tracings made in this manner the systolic pressure was read at 150, 152, 150, 148, 148 and 146.

It is my experience that by means of this visible indicating device the systolic blood-pressure may be read with the same accuracy as obtained when using the more complicated instruments. By thus eliminating the variation due to personal equation, which may be great or small, according to the observer's experience, blood-pressure readings may be standardized.

This device was originally designed for taking blood-pressures in children, which is very difficult owing to the small size of the arms and the difficulty in obtaining uniform results either by tactile or auscultatory methods. Where the arm is too small for use, the cuff may be applied to the leg. The leg readings in children are from 10 to 15 mm. mercury higher than in the arm. It can, however, be used just as readily in adults as in children and may be attached to any type of instrument, mercurial or aneroid.

Therefore this device possesses the following advantages:

1. Automatically and visibly indicates systolic and diastolic pressure.
2. It compares very closely in efficiency with the Erlanger instrument.
3. It enables one to eliminate the variation due to personal equation, which has been shown to be considerable in different observers.
4. It is of especial use in children in whom blood-pressure readings are difficult, since it permits the use of the leg when the arm is too small.

131 East Sixty-Seventh Street.

TUBERCULOSIS OF THE MESENTERIC GLANDS IN INFANTS AND YOUNG CHILDREN; ITS EFFECT ON ABSORPTION *

FRITZ B. TALBOT, M.D.
BOSTON

Our conception of tuberculosis in early childhood has been modified by the work of various investigators, whose experiments seem to show that the common channel of infection is not so much the air passages, as the intestinal mucosa. The tubercle bacilli may pass through the intestinal wall, be carried by the lymphatics to the mesenteric glands, and thence into the thoracic duct and blood stream. These views do not reject the older hypothesis that the bacilli are borne by dust into the lungs.[1] Von Behring[2] says that "the milk fed to infants is the chief cause of consumption," but he also says that it is absolutely important to keep a coughing consumptive away from a nursing infant.

Corner[3] finds tuberculous mesenteric glands in the majority of children on whom he has performed a laparotomy, and believes that a large percentage of children have such lesions. He explains this relative frequency as follows: There is a stage of digestion in which there is an abrupt change in the mechanical and physical processes at the time of transition from a rapidly moving liquid fecal material of the small intestine, to a more slowly-moving, solidifying fecal mass of the large intestine. The point of transition is at the ileocecal valve where there is a temporary pause and the bacterial flora have an opportunity to multiply rapidly Cultures taken here and at other parts of the intestinal canal have shown that there are many more bacteria in the ileocecal region than elsewhere. Nature has furnished a large amount of lymphoid tissue in this region to combat the organisms which are presumably in their most infectious stage. He assumes, therefore, that the ileo-cecal valve is the most likely place of entrance of the tubercle bacilli. The lymph-follicles of the small and large intestines also serve as points of entry.[4] The report of the Royal

---

*From the Children's Medical Service, Massachusetts General Hospital.

*Read in the Section on Diseases of Children of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

1. Riviere, Clive: Phthisis in Children, Lancet, London, Jan. 15, 1910.

2. Von Behring: Suppression of Tuberculosis, New York, 1904.

3. Corner: Tuberculosis of the Mesenteric Glands in Children; Its Nature and Treatment, Lancet, London, 1912, 1, clxxxii, 426.

4. Hof, Carl: Ueber primäre Darmtuberculose, Kieler Dissert., 1903; Hausemann, V.: Ueber Fütterungs Tuberculose, Berlin. klin. Wchnschr., 1903, No. 7, quoted from Von Behring (Note 2).

Commission on Tuberculosis[5] had twenty-nine cases of primary abdominal tuberculosis of which fourteen yielded the bovine type of bacillus, thirteen the human type and two were proved to contain a mixture of the bovine and human tubercle bacillus. There is, therefore, very good evidence that cows' milk is one of the common sources of contagion. Those familiar with babies and small children have frequently seen the variety of dirt that is picked up off the floor and swallowed, and cannot be surprised that the human type of tuberculosis also plays an important part.

The bacilli are carried by the lymphatics to the mesenteric lymph-nodes where they may lodge, eventually cause caseation and destroy the usefulness of these nodes and lymph-channels for digestion and absorption. If a sufficient number of the glands are destroyed, absorption through the lymphatics will be impossible and a dam will be placed across the channel through which certain foodstuffs gain entrance to the blood-stream and the rest of the body. It is necessary to review the manner in which the three great classes of foodstuffs, fats, carbohydrates and proteins, are absorbed.

Fats.—The majority of fats eaten by young children are in the forms of neutral fats. During the course of digestion they are hydrolized into fatty-acids and glycerin and, if they come in contact with alkaline intestinal juices, the fatty-acids combine with the salts and become soaps. They pass through the intestinal mucous membrane, and are taken up in the lymphatics as neutral fats, and are carried through the thoracic duct to the blood. If the lymph of the thoracic duct is collected, it shows only 60 per cent. of the fat which is absorbed from the intestine. It is not known, however, what happens to the other 40 per cent., but experiments have made it probable that it is not absorbed directly into the blood.[6] Any obstruction to the flow of lymph would, therefore, interfere with the absorption of fats.

Carbohydrates.—All carbohydrates are changed during digestion into monosaccharids, which are absorbed in the small intestine before they reach the ileocecal valve. The percentage of sugar in the thoracic duct does not increase during digestion of a meal rich in carbohydrates, while there is a definite increase in the percentage of sugar in the portal system.[7] These facts indicate that sugars are absorbed entirely by the blood of the portal system and not by the lymphatics.

Protein.—Numerous experiments make it probable that the proteins are taken up entirely by the blood of the portal system and that they are not absorbed through the lymphatics because during a meal rich in protein

5. Final Report, Part I, p. 13, London, 1911.
6. Starling in Oppenheimer's Handbuch der Biochemie des Menschen und der Tiere, iii, 2, pp. 225-6; Jena, 1909.
7. Starling: (See note 6) p. 241.

there is no appreciable increase in the flow of lymph or in the amount of protein which it contains.[8]

Physiology shows, therefore, that if the mesenteric lymph-nodes become diseased enough to block the entire flow of lymph, the protein and carbohydrate digestion and absorption will not be interfered with. At least 60 per cent. of the fat ingested may not be absorbed. The amount of interference with the absorption of fat would naturally depend on the number and location of the diseased glands and how completely they blocked the lymph-ducts and their numerous anastomoses.

Bergrün and Katz[9] describe the occurrence of fatty stools in tuberculous peritonitis and give a review of the literature up to 1891. Schmidt[10] describes a case of an 8-year-old girl, with intestinal and mesenteric tuberculosis, in whom all of the mesenteric glands proved to be caseous. The stools were diarrheal and contained a great deal of fat. Fr. Müller's[11] case, a child 4 years old with phthisis and caseation of the mesenteric and retroperitoneal lymph-nodes, had yellow-gray, mealy stools which showed under the microscope many fat crystals. These stools contained 42 per cent. of fat. Wietrand[12] performed metabolism experiments in a case of tuberculosis of the intestine and found that 35 per cent. of the fat ingested was lost in the feces. Wharton[13] says that in case of tabes mesentericus, "Absorption from the intestine becomes greatly diminished, the child greatly emaciated, the abdomen is distended, and an insatiable appetite, diarrhea and offensive stools are the chief symptoms."

I studied the cases reported in this paper with special reference to the digestion of fat, carbohydrates, and protein. The methods of examining the stools were those described in a previous paper.[14] The diet was modified according to the results of these examinations.[15] The findings may be summed up as follows: The cases were clinically typical cases of tuberculous peritonitis with positive von Pirquet skin reactions, in three of which the diagnosis was confirmed by laparotomy, and two more by autopsy. Meat and starch were digested in a normal manner, while fat appeared in the stools in a large excess in every case except one. In this instance, Case VI, it was fair to assume that the lymphatics were not entirely blocked. It was found that if fat was given in the food in large amounts, eventually the stools would change from a formed, apparently

8. Starling: loc. cit. Note 6, pp. 233-4.
9. Bergrün and Katz: Wien. klin. Wchnschr., 1891, No. 4, p. 858.
10. Schmidt: Ztschr. f. exper. Path. u. Therap., 1909-10, vii, 270.
11. Müller, Fr.: Untersuchung über Icterus. Ztschr. f. klin. Med., 1887, xii, 86.
12. Wietrand: Die Bedeutung des quantitativen Stoffwechselversuches, etc., Die Heilk., 1898-99, ii, 67.
13. Wharton: Osler's Modern Medicine. Phila. and New York, 1908, p. 823.
14. Archives of Pediatrics, 1911, xxviii, No. 2.
15. Morse and Talbot: Intestinal Indigestion in Children, Am. Jour. Med. Sc., June, 1910.

well-digested stool into a diarrheal, acid stool containing much mucus, and that signs of a secondary intestinal indigestion would appear. An example of this is Case X. This is presumably due to the fact that the excessive amounts of fat in the intestine are acted on by the intestinal bacteria whose action causes putrefaction. The stools often have the odor of rancid butter. As tuberculosis developed in Case VIII, the character of the stools changed. In the earlier stages fat was digested in the normal manner, and later a great deal was lost in the feces presumably because tuberculous mesenteric glands entirely blocked the lymphatic system.

The diet of these children should be regulated according to the stool-findings, and in all instances in which the disease has progressed far enough for the tuberculous processes to block the lymphatic system, fat should be removed entirely from the food, and the caloric needs of the child be supplied by carbohydrates and protein. When this is done the patient is usually much more comfortable, as was Case X, and the body has a better opportunity to combat the disease. In one instance, Case II, there was distinct improvement in the appearance of the child and she gained one-half pound in one month's time.

CASE REPORTS

CASE 1.—Catalina C., aged 9 years, M. G. H. O. P. D. No. 182906, entered the hospital Nov. 6, 1911, complaining that she was debilitated and anemic for one year. There was no known exposure to tuberculosis and she had never been sick aside from symptoms referrable to her stomach. She was brought in by the school nurse because she did not look well. The diet included macaroni, cocoa, pork and other foods of a similar nature. On questioning it was found that she had seven or eight stools a day.

Physical Examination.—This showed a fairly developed and poorly nourished, pale girl with a distended abdomen, diastasis of the recti, shifting dulness in the flanks and a fluid wave. There was slight general tenderness over the whole of the abdomen, but no masses were felt there. The circumference of the chest at the nipple was 60 cm. and of the abdomen over the umbilicus 63 cm. The physical condition was normal elsewhere except for moderate emaciation. The von Pirquet skin tuberculin test was positive. The diagnosis of tuberculous peritonitis (serous form) was made. Two days later examination of the stool under the microscope showed an excess of fats mostly in the form of fatty acids. Fat was removed entirely from the diet, and under careful dieting the stools dropped from 8 a day to 3 a day. The circumference of the abdomen on entrance was 63 cm. over the umbilicus, and one month later was 60 cm. and the fluid diminished in amount. When last seen there as no fluid wave, and the child reported that she was much better in every way.

CASE 2.—Carilda R., aged 20 months, M. G. H. O. P. D., No. 189977, entered the hospital March 12, 1912. One other child in the family died one month previously at the age of 4 years with "ascites." The child was breast fed for fifteen months and perfectly healthy until about one month prior to admission, at which time the mother noticed that the baby began to be limp and listless. Although she did not complain of pain she preferred to lie down most of the time. The abdomen became more and more prominent and recently tender. The stools had been hard, yellow and constipated. The diet consisted of whole milk, macaroni, rice, cereals and oranges.

Physical Examination.—This showed a poorly developed and nourished, pale child with a prominent, distended abdomen which was tympanitic throughout.

A large mass was felt extending across the upper part of the abdomen corresponding to the course of the transverse colon; this mass was tender. The von Pirquet skin tuberculin test was positive. The diagnosis of tuberculous peritonitis (fibrinous form) was made. Two days later the stool was recorded as loose, pinkish-brown and it contained large particles of orange pulp. Under the microscope there was no meat or starch, but there was a large excess of fat in the form of soaps. She was given a diet without fats and in the course of one month gained one-half pound. Although the general condition remained the same, the baby looked much better under careful dieting.

CASE 3.—Ralph W., age 4 years, M. G. H., No. 176350, was admitted to the hospital May 30, 1911. There was no known exposure to tuberculosis. As a baby, he was breast fed for three months and after that on the bottle. About six months prior to admission the stomach began to swell up and he complained of general abdominal pain, without vomiting. Four weeks ago the abdomen became considerable larger and the abdominal pains more marked. The bowels were slightly constipated.

Physical Examination.—This showed a poorly nourished, colored boy with an anxious expression. The muscles were flabby. The lungs were resonant, but contained many fine, medium and coarse râles. There was no change in the respiratory murmur. The level of the abdomen was a little higher than that of the thorax; it was tense but not tender. There were several masses the size of olives in the right lower quadrant, and no shifting dulness. There was an umbilical hernia which was excoriated. The diagnosis of tuberculous peritonitis and bronchitis was made and he was put on the "house diet" of the children's ward. This consisted of meat, vegetables, whole milk, bread, butter, cereals and cooked fruits. The stool was small, dark greenish-brown and semi-solid, with a moderate amount of mucus intermixed. Under the microscope it showed a moderate excess of fat in the form of soaps. There was no meat or starch. Eleven days later the stool was medium sized, light brown and reacted acid to litmus paper. Under the microscope it showed a large excess of fat in the form of soaps. Four days later the fats were partially removed from the diet, i. e., skimmed milk was given in place of whole milk and butter, and the stools showed very much less fat. He was discharged to his parents ten days later with a practically normal stools.

CASE 4.—William C., aged 2 years, M. G. H., No. 176419 was admitted Jan. 2, 1911. There was no family history of tuberculosis. The child was breast fed for sixteenth months and for the last four months had been eating milk, egg and a little bread. About three months prior to admission it was noticed that he was getting thin and "wabbled" as he walked. One month latter the abdomen began to increase in size and for the past ten days had been tender. No cough or vomiting; bowels regular.

Physical Examination.—This showed a poorly developed and nourished boy who was irritable and frightened; his muscles were flabby and more or less atrophied. The level of the abdomen was higher than that of the thorax; it was rigid, tense and somewhat tender. There was shifting dulness and a fluid wave. The umbilicus was reddened, indurated and with superficial excoriations; hard masses extended from it for about 4 cm. in all directions. There were also masses, the size of which were not reported, in both the left and right lower quadrants. The physical examination otherwise was normal. The diagnosis of tuberculous peritonitis (serous type) was made and he was given the "house diet" of the children's ward —meat, vegetables, whole milk, bread, butter, cereals and cooked fruit. The stool was large, yellow, firm and sour, the scraped surface being greasy. It contained no blood or mucus, but under the microscope there was a moderate excess of fat in the form of soaps. Two days later the umbilicus suddenly began to discharge a stream of pus which became fecal in character. This was opened and found to be a large, extra-peritoneal abcess. The diet was regulated as in Case 3.

CASE 5.—Evelyn R., aged 4 months, M. G. H., No. 173935, was admitted Jan. 12, 1911. The mother was at a sanatorium being treated for tuberculosis, and very little was known about the baby except that she had been fed mostly on the bottle.

Physical Examination.—This showed a poorly developed and nourished child with a facial paralysis on the right side of the face, and a discharge from the right ear underneath which there were several large lymph-nodes. The lungs were normal except for a few fine râles in both backs. The abdomen was large and somewhat resonant, with a protruding umbilicus. There was no rigidity, spasm or masses. The physical examination was otherwise negative. She was put on a formula which figured 2.75 per cent fat, 6.00 per cent sugar, and 1.50 per cent. protein; 4½ ounces every three hours, seven feedings. The stool two days later was small, greenish-yellow, with no curds or mucus. The reaction was alkaline and under the microscope it showed a large excess of fats. The von Pirquet skin tuberculin test was negative. Four days later after the food had been regulated according to the stool findings, i. e., the fats were reduced to 2.00 per cent., the stool was reported as small, normal in appearance, of alkaline reaction and containing under the microscope a small amount of fat in the form of soaps. The baby died January 22, and the necropsy report was as follows:

Necropsy.[16]—Esophagus and stomach on section are normal. The large intestine is not remarkable. The walls of the small intestine are generally pale, but in several places show small, dark reddish areas dotted over with tubercle-like points. One of these areas is situated in the region of the ileocecal valve and the others, beginning at a point about 30 cm. above the ileocecal valve, are scattered along the ileum. The mesenteric lymph-nodes generally are enlarged and studded with smaller and larger yellowish caseous masses. These caseous glands are in close relation with the dark reddish tubercle-studded area in the wall of the ileum. On section the mucosa of the ileum shows, opposite the dark reddish areas mentioned, small losses of substance with reddish, rather ragged margins and bases, the latter showing tubercle-like points. There are about a dozen of the ulcers lying transversely and the largest is about 1 cm. in greatest dimension.

The retroperitoneal lymph-nodes in the region of the pancreas and lesser omentum are enlarged and show many minute to small yellowish caseous masses.

Diagnosis.—The anatomic diagnosis was as follows: Tubercular ulcers of the ileum; tuberculosis of the mesenteric and retroperitoneal glands; miliary, focal and caseous pneumonia of the lungs; tuberculosis of the liver, spleen and adrenals; solitary tubercle of pia-arachnoid; solitary tubercle of the aorta; streptococcus septicemia.

CASE 6.—Charles P., aged 3 years, M. G. H., No. 177457, was admitted July 27, 1911. There was no history of tuberculosis. The baby was breast fed for one year, at which time he had measles followed by bronchopneumonia. After that the diet was the ordinary diet of the age. For the previous three months he had complained of pain in the epigastrium. The abdomen was swollen, especially in the upper part. He had lost considerable weight but had never vomited. The bowels were regular.

Physical Examination.—This showed a well developed and nourished child with the appearance of perfect health. The abdomen was very prominent, tympanitic, with a general tenderness and spasm. There was no shifting dulness, no fluid wave or masses. The physical examination was otherwise normal. Two days later an indefinitely defined mass was felt across the upper part of the abdomen just above the umbilicus. The von Pirquet tuberculin skin test was positive. The diagnosis of tuberculous peritonitis (fibrous form) was made and he was given the "house diet." The stool was normal in appearance and contained no blood or pus. Under the microscope it showed a small amount of fat in the

16. This autopsy report was kindly given to me by Dr. Oscar Richardson of the Massachusetts General Hospital.

form of soaps. This was probably not in excess. After the child became used to the hospital the mass in the upper part of the abdomen was made out and corresponded more or less to the position of the transverse colon. A laparotomy performed August 4 showed a dry, tuberculous peritonitis with tubercles and numerous adhesions throughout the abdominal cavity. No observation was made of the condition of the mesenteric glands. Presumably these glands were more or less affected. The examination of the stool, however, makes it probable that the lymphatic system was not entirely blocked because there was not an excess of fat in the stools. It was not necessary to modify the diet in this instance because the boy digested and absorbed his food in a normal manner.

CASE 7.—Clarence N., aged 15 months, M. G. H., No. 178582, was admitted September 20, 1911. No known exposure to tuberculosis. He had always been bottle fed. Had had a slight cough for three weeks, and had been really sick for the previous week. The bowels and urine had been apparently normal. He breathed very rapidly and vomited practically all food. The physical examination showed that the lungs were resonant throughout and normal, except for many coarse and fine râles especially at the left base. The abdomen was full, distended and tympanitic. There was a slight general spasm but no masses or shifting dulness. The physical examination was otherwise negative. The von Pirquet skin tuberculin test was positive two days later. A tentative diagnosis of tuberculous bronchopneumonia was made and he was given a diet of whole milk and cereals. There was no more vomiting. The stool on October 10 was yellowish-green with considerable fine mucus, but no curds, pus or blood. Under the microscope there was a large excess of fat in the form of soaps. On October 15, after the diet had been regulated according to the stool-findings, i. e., skimmed milk was given in place of whole milk, the stool was greenish-yellow with a small amount of mucus and under the microscope a slight excess of soaps; it contained no tubercle bacilli.

The baby died October 21, and the necropsy was as follows:

Necropsy.[17]—Esophagus, stomach and small intestine on section are normal. The large intestine shows in its mucosa in the region of the junction of the cecum and first portion of the ascending colon an oval loss of substance with fairly smooth, rounded, slightly irregular margins which descend rather abruptly and deeply to a rather thin, grayish base which is slightly rough and irregular in its central portions. The large intestine elsewhere is not remarkable. The ulcer situated near the mesentery and the mesentery in this region show several small nodes, and a short distance from this a large mass of mesenteric nodes about 5 by 4 by 4½ cm. over all. On section of the large mass much caseo-pus is yielded, and a cover-glass specimen from it shows numerous typical tubercle bacilli. The section surfaces of the glands show discrete and confluent smaller and larger yellowish, homogeneous areas. The mesenteric glands elsewhere are visible but show no good evidence of any tuberculosis. The retroperitoneal glands are not remarkable. The lungs and other organs are normal.

Diagnosis.—The anatomic diagnosis was tubercular ulcer of the large intestine; tuberculosis of the mesenteric lymph-nodes; miliary tuberculosis of the spleen and liver; streptococcus septicemia.

CASE 8.—Samuel F., aged 3 years, M. G. H., No. 177995 and No. 179067, was admitted Aug. 28, 1911. No known exposure to tuberculosis. For the previous six weeks the patient had been failing; had had slight cough and fever all the time, and for the past three days his abdomen had gradually increased in size. He had lost a great deal of weight, and his appetite had been very poor.

Physical Examination.—This showed a poorly developed and emaciated child with distinct pallor. His abdomen was large and very prominent, the level being above that of the thorax. It was tympanitic throughout, and had a slight

17. Autopsy by Dr. Richardson, Massachusetts General Hospital.

"doughy" feeling. There were no masses or shifting dulness. The superficial veins were very much distended. While taking the "house diet" the stools were brown, homogeneous and formed; there was no blood, pus or mucus and under the microscope no excess of fat or starch. The von Pirquet tuberculin skin test was mildly positive. The physical examination was otherwise normal.

Between September 18 and October 30 he had diphtheria October 30 the physical examination was practically the same as at first noted. The stool, however, was brown, full and reacted alkaline to litmus paper. It contained many coarse, undigested particles, and under the microscope showed a large excess of fat in the form of soaps. The fat was then removed from the food, and there was marked improvement in the character of the stools. November 2 a laparotomy showed a small amount of fluid pus and on exploration a large mass of lymph-nodes the size of marbles in the mesentery. The intestines were adherent to the peritoneum just above the incision. The surgeon made a diagnosis of tuberculosis of the peritoneum and mesenteric glands.

CASE 9.—Thomas B., aged 5 years, M. G. H., No. 180665, was admitted Jan. 23, 1911. There was no known history of tuberculosis. The child was brought in because he had been found pale and thin for one week. The abdomen was much enlarged and tender. The appetite was good until the previous day. The diet was not good, as he ate candy, pie, cake and other foods from the table.

Physical Examination.—This showed a well developed and fairly nourished, pale boy. The abdomen was full, distended and tympanitic throughout. There was a slight tenderness on pressure and marked shifting dulness in the flanks, but no fluid wave. The von Pirquet skin tuberculin test was markedly positive. After he had been on the "house diet" three days, the stool was dark brown, soft, very foul; no neutral fats or fatty acids; there was a large excess of soaps. And again, on January 31, after the fat had been removed from the food, the stool was pasty, pale and still contained a large excess of fat.

CASE 10.—I. T., a girl, 4½ years of age, was first seen Aug. 24, 1911. There was no known history of tuberculosis. In January, 1911, she had measles and did not recover satisfactorily; the Moro skin test was done and found to be positive. At that time she had a little cough and was examined by four physicians in a sanitaria for tuberculosis. These men could find no evidence of tuberculosis of the lungs. The radiograph of the chest also showed no sign of tuberculosis. Her appetite was never hearty. About the first of July she developed a diarrhea which persisted. She lost a good deal of weight. On examination she was found to have a positive von Pirquet skin tuberculin test, and the stools under the microscope showed a large excess of fats.

Physical Examination.—This was normal except that the abdomen was slightly prominent, especially in the lower part. In the right iliac fossa there was a mass the size of a small peach and just to the left and below the umbilicus was a second mass the size of an olive. There was a slight indefinite resonance all over the abdomen, but no spasm of tenderness. A few days later, on a diet free from fat, the stool was large, greenish, with many flecks of mucus. Under the microscope there was a slight excess of fat, no starch or meat fibers. There was a very large number of pus cells. A stained smear showed no tubercle bacilli.

The feces were injected into a guinea-pig which was killed six weeks later and found to contain many tubercle bacilli.

Whenever even smaller quantities of fat were given, the stools became more frequent, and had the odor of rancid butter, and there was marked distress. The coincident discomfort would destroy the appetite so that at times she would eat practically nothing. As long as the fat was removed entirely from her food she lived in comparative comfort up to the time of her death two months later.

311 Beacon Street.

PROGRESS IN PEDIATRICS

INDEX OF CURRENT PEDIATRIC LITERATURE

ANATOMY, PHYSIOLOGY AND HYGIENE

Sterilization of Milk Bottles with Calcium Hypochlorite. H. A. Whittaker and B. M. Mohler.
Am. Jour. Pub. Health, April, 1912.

Portable Outfit for Determination of Visible Dirt in Milk. F. O. Tonney.
Am. Jour. Pub. Health, April, 1912.

Medical Milk Commission and Its Relation to Public Health. C. A. Rhodes.
Jour. Med. Assn. Georgia, May, 1912.

Importance of Milk Stations in Reducing City Infant Mortality. A. E. Howard.
Boston Med. and Surg. Jour., May 23, 1912.

Physiologic Importance of the Normal Intestinal Flora. (Bedeutung der normalen Darmflora.) M. Klotz.
Berl. klin. Wchnschr., May 6, 1912.

Vaccination. S. S. Moody.
Jour. Tennessee Med. Assn., April, 1912.

Best Means of Combating Infant Mortality. A. Jacobi.
Med. Rec., New York, June 8, 1912.

Cool Beds for Infants. A. F. Hess.
Arch. Pediat., May, 1912.

Some Newer Aspects of Problem of Infant Hygiene. A. McAllister.
Arch. Pediat., May, 1912.

Examination of Country School Children. A. G. Fort.
Jour. Med. Assn. Georgia, May, 1912.

School Sanitation and Medical Inspection of Schools. E. E. Dorr.
Iowa Med. Jour., Des Moines, May, 1912.

Young Infants in Institutions. (Säuglingspflege.) S. Engel.
Therap. Monatsh., May, 1912.

Want of School Dentistry. G. Sheppard.
Med. Press and Circular, May 1, 1912.

BACTERIOLOGY AND PATHOLOGY

Bacteriologic Study of Milk Supply of Washington, D. C. J. J. Kinyoun and L. V. Dieter.
Am. Jour. Pub. Health, April, 1912.

Chvostek's Sign in Older Children. (Bedeutung des Fazialisphänomens jenseits des Säuglingsalters.) H. Neumann.
Deutsch. med. Wchnschr., April 25, 1912.

METABOLISM AND NUTRITION

Nutrition and Digestion of Infants. M. Ladd.
Arch. Pediat., May, 1912.

Variations in the Amount of Breast Milk and its Connection with the Fat Content. (Zur Physiologie der Milchsekretion. II.) H. Helbich.
Monatschr. f. Kinderh., 1912, x, No. 12.

Necessity for Individualizing Intervals between Breast Feeding during First Weeks of Life. (Zur Technik der Ernährung der Brustkinder in den ersten Lebenswochen.) H. Rietschel.
Jahrb. f. Kinderh., April, 1912.

Technic for Breast Milk Feeding during First Month. (Zur Technik der Ernährung der Brustkinder in den ersten Lebenswochen.) H. Rietschel.
Jahr. f. Kinderh., May, 1912.

Lime Balance of Bottle-Fed Infant. (Kalkbilanz eines künstlich genährten Säuglings.) A. Niemann.
Jahrb. f. Kinderh., May, 1912.

Minor Points in Infant Feeding. R. A. Benson.
Am. Jour. Obst., May, 1912.

Feeding of Children from Bottle to Puberty. E. Campbell.
Lancet-Clinic, May 4, 1912.

Duodenal Alimentation. W. G. Morgan.
Am. Jour. Med. Sc., May, 1912.

Infant Feeding at Ashfield Home for Infants. A. E. Mills.
Australian Med. Jour., April 13, 1912.

Some Common Disorders of Nutrition in Infancy. H. C. Cameron.
Clin. Jour., May 15, 1912.

Pathology of Nuclein Metabolism. (Gicht. Uratsteindiathese.) R. Schütz.
St. Petersb. Med. Ztschr., April 28, 1912.

Digestion of Wheat Flour and Oatmeal. (Ueber den differenten peptischen Abbau von Weizen und Hafer.) M. Klotz.
Monatschr. f. Kinderh., May, 1912.

Favorable Action of Forced Feeding on Exclusive Milk-Vegetable Diet in Children of the Exudative Diathesis Plus Mild Tuberculosis. (Zur Diätetik der Skrofulöse.) E. Moro.
Monatschr. f. Kinderh., May, 1912.

Favorable Action of Cod Liver Oil in Rachitis not Annulled by Long Heating to Boiling Point. (Zur Frage vom Wesen der günstigen Wirkung des Lebertrans bei Rachitis. Ist das wirksame Agens des Lebertrans ein Ferment?) J. A. Schabad and R. F. Sorochowitsch.
Monatschr. f. Kinderh., May, 1912.

Diet without Milk in Children with Spasmophilia. (Ueber die Durchführung einer milchlösen Kost bei Spasmophilie-Krampfbereitschaft.) W. Raabe.
Therap. Monatsh., May, 1912.

Surgery of Rachitis. H. L. Taylor.
Arch. Pediat., May, 1912.

Indicanuria. A. L. Benedict.
Arch. Diagnosis, April, 1912.

DISEASES OF THE NEWLY-BORN

Fever in New-Born Infant. E. Cautley.
Brit. Med. Jour., May 18, 1912.

Patent Duct of Botalli. (Sopra un caso di "morbo azzurro" da probabile persistenza del dotto arterioso do Botalli.) A. D. Piane.
Riforma med., April 20, 1912.

Umbilical Tumors Containing Uterine Mucosa or Remnants of Müller's Ducts. T. S. Cullen.
Surg., Gynec. and Obst., May, 1912.

Preventive Silver Nitrate Treatment of Umbilical Cord. (Om præventiv Lapisbehandling af Navlesnorsstumpen og Navlesaaret hos nyfødte.) F. Teilmann.
Hospitalstid., April 12, 1912.

Congenital Absence of Colon. J. Morton.
Brit. Med. Jour., May 18, 1912.

Congenital Absence of the Femur (Ueber angeborenen Femurdefekt.) O. Creite.
Deutsch. Ztschr. f. Chir., April, 1912.

Surgical Management of Developmental Defects in New-Born. H. Greenburg.
Wisconsin Med. Jour., May, 1912.

Importance of Laceration of Tentorium in the New-Born. (Bedeutung der Tentoriumrisse für das neugeborene Kind.) A. Bauereisen.
München. med. Wchnschr., May 7, 1912.

Benefit to be Expected from Operative Treatment of Congenital Coxa Vara. (Was kann man bei angeborener Coxa-vara auf blutig operativem Wege erreichen?) A. Mendler.
München. med. Wchnschr., May 14, 1912.

Study of Icterus Neonatorum by Means of Duodenal Catheter. A. F. Hess.
Am. Jour. Dis. Child., May, 1912.

ACUTE INFECTIOUS DISEASES

Practical Points in Diagnosis of Exanthemata and Allied Diseases. M. Young.
Practitioner, London, May, 1912.

Chorea and Infection. J. Roux.
Bull. Soc. de pédiat. de Paris, April, 1912.

Chorea. (Behandlung der Chorea minor.) J. Grober.
Deutsch. med. Wchnschr., May 2, 1912.

Use of Flexner Serum in Cerebrospinal Meningitis. E. G. Hynes.
Am. Jour. Obst., May, 1912.

Tuberculous Meningitis. F. M. Conlin.
West. Med. Rev., May, 1912.

Influenzal Meningitis. G. R. Pisek.
Am. Med., April, 1912.

Local Specific Treatment of Infections, with Special Reference to Epidemic Meningitis. S. Flexner.
Edinburgh Med. Jour., May, 1912.

Gangrene of Leg in Diphtheria. (Cas de gangrène embolique d'un membre consécutive à une angine diphthérique maligne.) E. C. Aviragnet, Blechmann and A. Huber.
Arch. de med. d. enf., May, 1912.

Prophylaxis of Measles, Mumps and Chicken-Pox. (La lutte contre les maladies benignes.) Nageotte-Wilbouchewitch.
Bull. Soc. de pédiat. de Paris, April, 1912.

Salvarsan and Measles: Clinical Note. L. L. Williams.
Boston Med. and Surg. Jour., May 16, 1912.

Prevention of Ophthalmia Neonatorum. R. C. Mackenzie.
Boston Med. and Surg. Jour., May 16, 1912.

Neuropathic Constitution an Etiologic Factor in Poliomyelitis. M. Neustaedter.
Arch. Diagnosis, April, 1912.

Prognosis in Acute Epidemic Poliomyelitis as to Life and Disability. J. V. Manning.
Arch. Diagnosis, April, 1912.

Diagnosis of Poliomyelitis with Misleading Symptoms. T. A. Williams.
Kentucky Med. Jour., May 1, 1912.

Is Acute Anterio-Poliomyelitis, or Infantile Paralysis Infectious, or Strictly a Sporadic Disease? C. E. Edgerton.
New Orleans Med. and Surg. Jour., May, 1912.

Scarlatinal Lesions in Adrenals and Pancreas. (La surrénalite et la pancréatite scarlatineuses.) L. Tixier and J. Troisier.
Arch. de méd. d. enf., May, 1912.

Inclusion Bodies in Blood of Scarlet Fever as Means of Differentiation Diagnosis. M. Nicoll, Jr., and A. W. Williams.
Deutsch. Arch. f. klin. Med., 1912, cvi, Nos. 3-4.

Streptococci in Scarlet Fever. (Streptokokken bei Scharlach.) P. Yungmann.
Arch. Pediat., May, 1912.

Otitis Media in Scarlet Fever. (Om otitis media perforativa vid scarlatina.) G. Holmgren.
Hygiea, Stockholm, May 11, 1912.

Prophylaxis of Scarlet Fever. F. H. Pierson.
Jour. Med. Soc. New Jersey, May, 1912.

Salvarsan in Scarlet Fever. (Ueber die Beeinflussung des Scharlachs durch intravenöse Salvarsaninjektionen.) R. Lenzmann.
Med. Klin., April 28, 1912.

Serotherapy of Scarlet Fever. (Behandlung des Scharlachs mit Scharlachstreptokokkenserum.) X. Lewkowicz.
Monatschr. f. Kinderh., 1912. x, No. 12.

Disinfection in and after Infectious Diseases. W. T. Connell.
Canada Med. Assn. Jour., May, 1912.

Distribution of Cases Among Several Producers in Epidemics of Non-Milk-Borne Infectious Disease. W. T. Carpenter.
Am. Jour. Pub. Health, April, 1912.

Pain in Appendix Region during Acute Infectious Diseases. (Dolori appendicolari nel decorso di malattie infettive acute.) J. Illuminati.
Gazz. d. osp., April 18, 1912.

TUBERCULOSIS AND SYPHILIS

Probable Effect of Control of Milk-Supply on Infantile Mortality from Tuberculosis. S. Délepine.
Lancet, London, May 25, 1912.

Congenital Tuberculosis. A Dietrich.
Berl. klin. Wchnschr., May 6, 1912.

Tuberculosis in Infants. (Zur Klinik der Säuglingstuberkulose.) Eichelberg.
Monatschr. f. Kinderh., 1912, x, No. 12.

Modification of Tuberculin Reaction during Acute Infectious Diseases in Children. (Beobachtungen über v. Pirquets Tuberkulinreaktion bei akuten Infektionskrankheiten bei Kindern.) W. J. Moltschanoff.
Jahrb. f. Kinderh., April, 1912.

Intradermal Tuberculin Reaction in Children. (La réaction de Mantoux en clinique infantile.) Combe.
Rev. med. de la Suisse romande, March, 1912.

Tuberculin Treatment of Tuberculosis in Children. (Erfahrungen über die Behandlung der Kindertuberkulose mit dem Koch'schen Alttuberculin.) J. Cronquist.
Jahrb. f. Kinderh., May, 1912.

Treatment of Tuberculous Glands of Neck in Children. W. G. Sutcliffe.
Practitioner, London, May, 1912.

Primary Mesenteric Tuberculosis. (Klinisches und Pathologisches zur primären Mesenterialtuberkulose.) H. Schloessmann.
Beitr. z. Klin. d. Tuberk., April, 1912.

Tardy Manifestations of Inherited Syphilis. (2 Tilfælde af formentlig Lues hereditaria tarda.) E. Clausen.
Ugesk. f. Læger, May 2, 1912.

Later Development of Children with Inherited Syphilis. (Die weiteren Entwicklungs- und Gesundheitsverhältnisse der mit Lues congenita behafteten Kinder.) J. Vas.
Jahrb. f. Kinderh., April, 1912.

GASTRO-INTESTINAL SYSTEM

Appendicitis in Childhood. E. Marvel.
Arch. Pediat., May, 1912.

Management of Appendicitis in Childhood. W. F. Campbell.
Am. Jour. Obst., May, 1912.

Abdominal Distress in Children Beyond Infancy. L. Kerr.
New York Med. Jour., June 1, 1912.

Epidemic Gastro-Enteritis. S. Sheill.
Practitioner, London, May, 1912.

Intestinal Indigestion: Some Considerations of Diagnosis. H. Barclay.
Arch. Diagnosis, April, 1912.

RESPIRATORY SYSTEM

Bronchiolitis and Bronchopneumonia in Young Children. E. Feer.
Med. Klin., April 21, 1912.

Empyema of Infancy. S. A. Blauner.
Am. Jour. Obst., May, 1912.

Influenza in Infants and Children. H. Schwarz.
Am. Med., April, 1912.

Effect of Cold Fresh Air on Blood-Pressure in Pneumonia of Children. J. Howland and B. R. Hoobler.
Am. Jour. Dis. Child., May, 1912.

CIRCULATORY SYSTEM

Anemia Associated with Rickets and Gastro-Intestinal Disturbances, Including Splenic Anemia in Children. H. T. Ashby.
Practitioner, London, May, 1912.

Blood of Infancy and Early Childhood. J. B. Manning.
Northwest Med., May, 1912.

Endocarditis not Entailing Valvular Defect. (Zur Frage der ohne Funktionsstörung im Sinne eines Herzfehlers verläufenden chronischen und recidivierenden Endocarditis der Mitralis und der Aortenklappen.) C. Amsler.
Cor.-Bl. f. schweiz. Aerzte, April 10, 1912.

NERVOUS SYSTEM

Epilepsy (Der Status epilepticus.) P. Jödicke.
Deutsch. med. Wchnschr., May 9, 1912.

GENITO-URINARY SYSTEM

Cystitis in Children. (Febbre intermittente da cistite nell-infanzia.) R. Petrucci.
Gazz. d. osp., April 28, 1912.

Nocturnal Urination. A. G. Miller.
Edinburgh Med. Jour., May, 1912.

Rupture of Kidney in Children. C. L. Gibson.
Am. Jour. Med. Sc., May, 1912.

OSSEUS SYSTEM

Coxa Vara and Consecutive Spontaneous Fracture. (Coxa flecta des adolescents et fracture spontanée justa-epiphysaire du col du fémur.) M. Gangolphe.
Lyon chir., May, 1912.

Diagnosis of Hip-Joint Disease from Displacement of Trochanter. (Hat die Prüfung der Roser-Nélaton'schen Linie diagnostischen Wert?) E. Schwarz.
Beitr. z. Klin. d. Tuberk., April, 1912.

Disturbances in Bone Growth from Deformity of Periosteum and Cartilage. (Störungen in der periostalen und endochondralen Knochenbildung). G. Joachimsthal.
Berl. klin. Wchnschr., April 22, 1912.

Ossification of Tuberosity of Tibia in Connection with Certain Disturbances in Children. (Untersuchungen über die Verknöcherung der Tubercositas anterior tibiæ mittelst Röntgenstrahlen, in Bezug auf einige Krankheiten der Jugend.) C. Licini.
Beitr. z. Klin. d. Tuberk., April, 1912.

Cysts in Bones. (Isoliert auftretende cystische und cystischfibröse Umwandlung einzelner Knochenabschnitte.) R. Stumpf.
Deutsch. Ztschr. f. Chir., April, 1912.

Congenital Acromegaly. (Fall von angeborener abnormer Grösse der Extremitäten mit einem an Akromegalie erinnernden Symptomenkomplex.) V. Salle.
Jahrb. f. Kinderh., May, 1912.

Rachitic Osteochondritis. M. Kassowitz.
Jahrb. f. Kinderh., May, 1912.

Straight and Crooked Legs. (Das Gesetz von der Umformung der Beine und die X-Beine unserer Frauen.) K. Francke.
München. med. Wchnschr., April 23, 1912.

Relation of Scoliosis to School Life. R. W. Lovett.
Lancet-Clinic, May 4, 1912.

Fracture of Greater Tuberosity of Humerus by Muscular Action in Child. W. P. Coues.
Boston Med. and Surg. Jour., May 30, 1912.

SKIN AND APPENDAGES

Management of Infantile Eczema. A. J. Markley.
Colorado Med., May, 1912.

Skin Reaction in Serum Sickness. (Zur Frage der Kutanreaktion.) F. Bauer.
Monatschr. f. Kinderh., 1912, x, No. 12.

EYE, EAR, NOSE AND THROAT

Adenoids: Their Effects on General System. J. J. King.
Pennsylvania Med. Jour., May, 1912.

What Adenoids Really Mean to Children. M. Yearsley.
Practitioner, London, May, 1912.

Tonsils in Childhood. J. G. Wilson.
Am. Jour. Dis. Child., May, 1912.

Tonsillotomy or Tonsillectomy. E. Danziger.
New York Med. Jour., June 1, 1912.

Tonsillar and Adenoid Affections of School Children, Their Dangers and Proper Correction. F. E. V. Shore.
Iowa Med. Jour., Des Moines, May, 1912.

Tonsillitis. C. L. Nicholas.
Kentucky Med. Jour., May 15, 1912.

Tonsillectomy; Consideration of Its Complications. C. W. Richardson.
Washington Med. Ann., May, 1912.

Epidemic of Throat Infection with Glandular Enlargement. F. Van Der Bogert.
Arch. Pediat., May, 1912.

Comparison of Streptococci from Milk and from Human Throat. E. C. Stowell and C. M. Hilliard.
Am. Jour Dis. Child., May, 1912.

Pathology of Deaf Mutism. G. H. Makuen.
New York Med. Jour., June 8, 1912.

Complication of Influenza Affecting Ear, Nose and Throat of Children. S. Oppenheimer.
Am. Med., April, 1912.

Passage of Hexamethylenamin into Cerebrospinal Fluid. (Zur Kenntnis der Urotropin-Sekretion und Resorption im Liquor cerebrospinalis und die Bedeutung derselben für die Hydrocephalus-Frage.) E. Weinrich.
Monatschr. f. Kinderh., May, 1912.

SURGERY AND GYNECOLOGY

Advantages of Transverse Tracheotomy. (Vorteile des queren Luftröhrenschnittes bei Kindern und das Tracheotomieren auf liegender Intubationskanüle.) H. Keiner.
Monatschr. f. Kinderh., 1912, x, No. 12.

Artistic and Mathematically Accurate Method of Repairing Defect in Cases of Harelip. J. E. Thompson.
Surg., Gynec. and Obst., May, 1912.

Fistulas and Cysts in the Neck. (Halsfisteln und Cysten.) R. Wenglowski.
Arch. f. klin. Chir., June 1, 1912.

Operations for Hare-Lip and Cleft Palate. (Ueber Uranoplastik.) J. Schoemaker.
Arch. f. klin. Chir., June 1, 1912.

Correction of Rachitic Deformity by Osteotomy and Fixation by Screws. (Redressement des déviations rachitiques invétérées des membres supérieurs et inférieurs par Osteotomie et Osteosynthèse-fixateur de Lambotte.) A. Machard.
Rev. méd. de la Suisse romande, April, 1912.

Experiences with 100 operations to correct cleft palate: Uranoplasty. (Erfahrungen bei 100 Gaumenspaltoperationen mit technischen Mitteilungen.) C. Helbing.
Berl. klin. Wchnschr., May 20, 1912, No. 21.

MISCELLANEOUS

Thickness of Adipose Layer in Children. (Dicke des Fettpolsters bei Kindern.) H. Neumann.
Jahrb. f. Kinderh., April, 1912.

Fever from Saline Infusion. (Zur Frage der Infusion physiologischer Kochsalzlösung.) A. Sippel.
Deutsch. med. Wchnschr., April 25, 1912.

Roentgenoscopy of Congenital Hernia of the Diaphragm. (Die congenitale Hernia diaphragmatica im Foramen Morgagni und ihre Röntgendiagnose.) E. Waelli.
Arch. f. klin. Chir., April 27, 1912.

First Woman Practitioner of Midwifery and Care of Infants in Athens, 300 B. C. G. T. McMaster.
Am. Med., April, 1912.

Preservation and Restoration of Tendon Function. W. L. Brown.
Texas State Jour. Med., May, 1912.

Children with a Tail. (Zur Kenntnis des geschwanzten Menschen.) G. Schwarz.
München. med. Wchnschr., April 23, 1912.

Portable Incubator. (Tragbare Convense zum Transport von Frühgeborenen.) E. Welde.
Jahrb. f. Kinderh., May, 1912.

Roentgentherapy of Hypertrophy of the Thymus. (Le traitement roentgénien de l'hypertrophie du thymus. Ses indications comparées à celles de la thymectomie.) C. Regaud and R. Crémieu.

Cat and Transmission of Disease. C. A. Osborne.
Chicago Med. Recorder, May 15, 1912.

Case Report of Quadruplets. C. T. Ricketts.
Kentucky Med. Jour., May 1, 1912.

Similar Cases of Extreme Ages. J. Y. Welborn.
Kentucky Med. Jour., May 1, 1912.

Deformities of Children. W. A. McKenney.
Kentucky Med. Jour., May 1, 1912.

Heredity of Deafmutism and Importance of Heredity for Pathology. (Om den konstitutionella döfstumhetens ärftlighetsförhållanden samt några ord om ärftlighetsforskningens betydelse för sjukdomsläran.) H. Lundborg.
Upsala Läkaref. Förh., March 16, 1912.

Pathology of the Gait in Children. (Störungen des Bewegungs-Apparates im Kindesalter.) C. Sarfels.
St. Petersb. med. Ztschr., April 28, 1912.

Infant Consultations. (La consultation obstétricale de nourrissons.) E. Bonnaire.
Presse méd., May 8, 1912.